NEUROPHYSIOLOGICAL MECHANISMS IN THE DEVELOPMENT OF EXTERNALIZING BEHAVIOR PROBLEMS IN YOUNG CHILDREN

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To former, current, and future children.

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Externalizing behavior problems including aggression, inattention/hyperactivity, oppositionality, and conduct problems are prevalent and burdensome. The present report describes two studies that examined neural mechanisms in the development of externalizing problems in very early childhood when self-regulation skills are rapidly developing and potentially most malleable. Study 1 ($N = 27$) and Study 2 ($N = 64$) examined neural functioning in relation to self-regulation and externalizing problems in 2 1/2- to 3 1/2-year-old children. Event-related potential (ERP) components and electroencephalography (EEG) assessed neurophysiological functioning during oddball and go/no-go tasks. A subset of the children were assessed with EEG measures at multiple occasions at 6 month intervals. Because deficient self-regulation is considered to reflect an underlying phenotype of externalizing behavior, we examined neurophysiological risk markers in relation to behavioral measures of self-regulation (inhibitory control and sustained attention) and questionnaire measures of externalizing behavior problems.

Several neural markers were associated with self-regulation deficits and externalizing behavior problems: smaller oddball P3b amplitudes, longer oddball P3a latencies, too small *or* too large no-go N2 amplitudes, left frontal asymmetry, and less frontal alpha power. Less frontal alpha power, thought to reflect task-inefficient/excess neural processing, showed the

greatest predictive value among these markers, and predicted relative increases in externalizing problems over time with moderate accuracy. Findings suggest that future studies should examine a new model of no-go N2 amplitudes in relation to inhibitory control whereby there may be an optimal range of inhibitory processing. Too little inhibitory processing may reflect poorer inhibitory control and too much may reflect the recruitment of excess/inefficient inhibitory resources. Future studies should examine how to ensure the validity of neurophysiological data while reducing lost EEG data due to artifacts and poor behavior performance in order to maximize generalizability and clinical utility. In sum, longitudinal studies of neural functioning in relation to behavior in early childhood have the potential to greatly advance our understanding of mechanisms in the development of self-regulation and behavior problems.

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Abbreviations

ACC	**A**nterior **C**ingulate **C**ortex
ADHD	**A**ttention **D**eficit **H**yperactivity **D**isorder
AUC	**A**rea **U**nder the **C**urve
CBCL	**C**hild **B**ehavior **C**heck**l**ist
CD	**C**onduct **D**isorder
CI	**C**onfidence **I**nterval
DLPFC	**D**orso**l**ateral **P**ref**f**rontal **C**ortex
ECBI	**E**yberg **C**hild **B**ehavior **I**nventory
EEG	**E**lectro**e**ncephalo**g**raphy
ERP	**E**vent-**R**elated **P**otential
EXT	**Ext**ernalizing Behavior Problems
FIML	**F**ull **I**nformation **M**aximum **L**ikelihood
fMRI	**f**unctional **M**agnetic **R**esonance **I**maging
IFG	**I**nferior **F**rontal **G**yrus
IOM	**I**nstitute **O**f **M**edicine
MFG	**M**edial **F**rontal **G**yrus
MRI	**M**agnetic **R**esonance **I**maging
NIH	**N**ational **I**nstitutes of **H**ealth
NIMH	**N**ational **I**nstitute of **M**ental **H**ealth
NRC	**N**ational **R**esearch **C**ouncil
ODD	**O**ppositional **D**efiant **D**isorder
OFC	**O**rbito**f**rontal **C**ortex
PCA	**P**rincipal **C**omponent **A**nalysis
PFC	**P**ref**f**rontal **C**ortex

ROC	**R**eceiver **O**perating **C**haracteristic
RDoC	**R**esearch **Do**main **C**riteria
SEM	**S**tructural **E**quation **M**odeling
SFG	**S**uperior **F**rontal **G**yrus
TMS	**T**ranscranial **M**agnetic **S**timulation
VLPFC	**V**entrol**l**ateral **P**ref**f**rontal **C**ortex

"Baseball was made for kids, and grown-ups only screw it up."

Robert Granville "Bob" Lemon

"Toutes les grandes personnes ont d'abord été des enfants, mais peu d'entre elles s'en souviennent." [All grown-ups were children first, but few remember it.]

Antoine de Saint-Exupéry, *Le Petit Prince*

Chapter 1

Introduction

1.1 Overview

The focus of the present study is on externalizing behavior problems such as aggression and attention problems, which produce major costs to society, and which are treatment-resistant when fully established. The present research seeks to identify neural mechanisms in the development of externalizing behavior problems and deficits in self-regulation among young children using event-related potential (ERP) recordings of neural activity from electroencephalography (EEG).

1.1.1 Why We are Conducting the Present Study

Externalizing behavior problems are common and are an enormous cost to individuals, families, and society. The median prevalence estimate of disruptive behavior disorders among children and adolescents in the general population from studies between 1993 and 2005 was about 6% (Costello, Egger, & Angold, 2005), with limited geographical variation in prevalence (Erskine et al., 2013). One example of an externalizing disorder is attention deficit hyperactivity disorder (ADHD). In 2000, ADHD was estimated to cost 31.6 billion

dollars in the United States (Birnbaum et al., 2005). It is well established that externalizing problems in young children strongly predict later, more severe problems such as conduct disorder, ADHD, and substance abuse. Although fully-developed externalizing disorders are often difficult to treat, prevention efforts targeted at disorders or precursors to disorders in younger children have been more efficacious and cost-effective in altering later trajectories than in those in older children (NRC & IOM, 2009). Understanding the early neural mechanisms in the development of externalizing problems may be crucial for earlier identification and prevention.

Early brain structure and functioning has successfully predicted, with sensitivity and specificity, the development of later dyslexia (Molfese, 2000) and autism (Elsabbagh et al., 2012; Stoner et al., 2014; Wolff et al., 2012) at ages before behavioral measures are useful. Therefore, it is likely that brain functioning could also predict the development of externalizing problems from a very early age. Establishing the early neural mechanisms in the development of externalizing problems may also help better identify early behavioral measures of risk, such as inhibitory control deficits. The importance of studying the neural mechanisms and precursors of the development of externalizing behavior problems is obvious. There is a striking lack of research, however, examining the neural basis of the development of externalizing behavior in early childhood. The importance of understanding the neural basis of the early development of externalizing behavior and the general lack of relevant research motivated the presented the studies.

1.1.2 Specific Aims

Neural biomarkers have been linked frequently to externalizing problems in adolescents and adults (Barry, Johnstone, & Clarke, 2003). What is not empirically established is (a) whether neural biomarkers in young children predict the early development of externalizing behavior problems, and (b) what are the best early behavioral indicators reflecting these neural deficits in the development of externalizing problems. Prior research suggests that we would find such links in early childhood, but this must be empirically tested. Based on these questions, the present research examines two specific aims: (1) Identify early neural biomarkers of the development of externalizing problems, and (2) Identify mechanisms underlying the development of externalizing problems. Better understanding of the developmental mechanisms of externalizing problems may improve early identification of children at risk for developing externalizing disorders and early intervention and prevention.

1.2 Definitions of Terms

The present study examines behavior problems from the perspective of developmental psychopathology, the study of "the interplay among the biological, psychological, and social-contextual aspects of normal and abnormal development across the lifespan" (Cicchetti, 2006, p. 1). Three dimensions of behavior problems can be used to summarize many cases of psychopathology: externalizing problems, internalizing problems, and disordered thought processes (Bates, Schermerhorn, & Petersen, 2014). The present study focuses on the mechanisms in the development of externalizing problems.

1.2.1 Externalizing Behavior Problems

Externalizing behavior problems represent a wide range of acting-out behavior problems. Externalizing behavior problems were so named because they reflect the child acting out on his or her external environment (Achenbach, 1966).[1] In contrast, internalizing problems, such as depression and anxiety, were originally named because of the seemingly inner-directed nature of one's conflicts. Externalizing problems consist of many different behaviors, including aggression, rule breaking, delinquency, attention deficits, hyperactivity, impulsivity, disruption, undercontrol, antisocial behavior, conduct problems, substance use, noncompliance, oppositionality, and defiance. Externalizing problems represent a useful grouping of these acting-out behaviors because it is a parsimonious description of many behaviors that tend to co-occur and appear to have similar etiologies (Bates et al., 2014; Liu, 2004; Olson, Bates, Sandy, & Lanthier, 2000). Moreover, externalizing problems in young children are meaningful—externalizing problems show a similar factor structure over time and individual differences in externalizing problems are relatively stable, even as early as 8 months of age (Lorber, Del Vecchio, & Slep, in press).

1.2.2 Self-Regulation

In the present study, aspects of self-regulation are examined as possible endophenotypes or intermediate phenotypes of the influence of neural mechanisms in the development of externalizing behavior problems. Self-regulation is a broad construct encompassing physiological, attentional, cognitive, emotional, and behavioral regulatory processes that promote

[1] According to Achenbach (1966, p. 10), "The [Externalizing/Internalizing] label is not intended to carry dynamic implications. It means only that the symptoms at the Externalizing end describe conflict with the environment, while those at the other end describe problems within the self."

adaptive or goal-directed behavior (Berger, 2011; Calkins & Fox, 2002). Self-regulation is conceptually related to the constructs of effortful control and executive function, which reflect differences in research tradition rather than real construct differences (Zhou, Chen, & Main, 2012). Executive function is a construct from the neuropsychological literature that emphasizes the higher-order cognitive processes involved in self-regulation that are related to the functioning of the prefrontal cortex (PFC; Zelazo, Carlson, & Kesek, 2008), including working memory, inhibition, and set shifting (Best & Miller, 2010). Effortful control, on the other hand, is a multidimensional construct from the temperament literature consisting of "the efficiency of executive attention, including the ability to inhibit a dominant response, to activate a subdominant response, to plan, and to detect errors" (Rothbart & Bates, 2006, p. 129). The present study focuses on the self-regulatory processes of inhibitory control and sustained attention.

1.2.2.1 Inhibitory Control (or Response Inhibition)

Inhibitory control is defined as "the ability to inhibit responses to irrelevant stimuli while pursuing a cognitively represented goal" (Carlson & Moses, 2001, p. 1033). Inhibitory control is conceptually similar to response inhibition, or the capacity to withhold an automatic or prepotent response (Cragg, Fox, Nation, Reid, & Anderson, 2009). As such, response inhibition or inhibitory control is important for the willful control of behavior and impulses, and it promotes goal-directed (Luna & Sweeney, 2004) and adaptive social behavior (for a review, see Carlson & Moses, 2001). Moreover, deficits in response inhibition have been observed in childhood disorders, such as ADHD (for reviews, see Johnstone, Barry, Markovska, Dimoska, & Clarke, 2009; Oosterlaan, Logan, & Sergeant, 1998) and substance-use disorders

(for a review, see Ivanov, Schulz, London, & Newcorn, 2008). Inhibitory control deficits are considered an intermediate phenotype of externalizing problems (Gagne, Saudino, & Asherson, 2011; Patrick, Venables, Yancey, Hicks, Nelson, & Kramer, 2013) and ADHD (Barkley, 1997; McAuley, Crosbie, Charach, & Schachar, 2014). A meta-analysis found that inhibitory control has a medium effect size (commission errors: Hedge's $g = 0.49$; omission errors: $g = 0.59$) in relation to ADHD (Wright, Lipszyc, Dupuis, Thayapararajah, & Schachar, 2014).[2] McAuley, Crosbie, Charach, and Schachar (2014) found that inhibitory control deficits distinguished children with ADHD from typically-developing children, and that inhibitory control deficits persisted even when ADHD symptoms did not. In other words, compared to the behavioral symptoms in the current diagnostic criteria, inhibitory control likely reflects a more stable phenotype that is closer to the biological basis of externalizing behavior.

1.2.2.2 Sustained Attention

Attentional regulation reflects the ability to shift and focus attention as needed for adaptive behavior (Eisenberg, Smith, Sadovsky, & Spinrad, 2004), and involves many different functions including attentional engagement/disengagement, sustained attention, and attentional focusing. The regulation of attention is important because what we attend to influences what we think, feel, and do. As a result, attentional regulation has been conceptualized as part of a larger self-regulatory system (Rueda, Fan, et al., 2004), and is typically operationalized as three different attentional regulatory systems: alerting, orienting, and executive attention (Rothbart, Sheese, Rueda, & Posner, 2011). An important

[2]Hedges' g is a variation of Cohen's d that corrects for biases due to small sample sizes (Hedges & Olkin, 1985).

part of the alerting system of attention regulation is sustained attention (Yin et al., 2012), which is defined as "the ability to mobilize and maintain selectivity and concentration" (Ruff, Capozzoli, & Weissberg, 1998, p. 454).

1.3 Literature Review

1.3.1 Topics and Purposes of the Literature Review

The purpose of the present literature review is to describe and synthesize findings examining the brain structure and function involved in self-regulation and externalizing-related phenotypes. The review incorporates different measures of neural structure (e.g., magnetic resonance imaging) and function (e.g., functional magnetic resonance imaging, electroencephalography, event-related potentials, magnetoencephalography, positron emission tomography, transcranial magnetic stimulation). Where possible, the review focuses on studies of children. For interpreting the present study in the context of findings from previous studies, we present age ranges of participants in previous studies. For studies that reported the mean and standard deviation of ages rather than a range of ages, a range was calculated as: mean $\pm$ 1 standard deviation.

A review of the neurobiological correlates of self-regulation and externalizing problems warrants several notes of caution. First, in addition to the enormous difficulty and complexity of localizing psychological processes to particular brain structures (Brett, Johnsrude, & Owen, 2002), there are theoretical and philosophical reasons to interpret localization studies with caution. It is generally thought that complex psychological and behavioral processes are not limited to specific, localized brain structures, but rather represent the connectivity

and interactions among many neurons, neurotransmitters, and brain structures, in addition to the complex interactions of many other biological processes and processes external to the individual. Although the current review discusses some of the key brain structures implicated in various self-regulatory processes, the brain regions mentioned depend on their connectivity with other structures, and the regions are integral to more functions and processes than solely those self-regulatory processes detailed here. Moreover, self-regulation skills likely depend on basic skills such as language skills (Petersen, Bates, D'Onofrio, et al., 2013; Petersen, Bates, & Staples, 2015), working memory (Wolfe & Bell, 2004), and their associated neurobiological systems. The goal of the current review is, therefore, to provide an additional level of analysis from which to determine the mechanisms in the development of self-regulation and externalizing problems.

1.3.2 Description and Critique of Literature

1.3.2.1 Task Paradigms

1.3.2.1.1 Inhibitory Control. Most of the neurophysiological research on inhibitory control or response inhibition uses tasks such as go/no-go or stop-signal tasks. In a typical go/no-go paradigm, participants are instructed to respond to some (go) stimuli, but not to other (no-go) stimuli, which induces a response conflict. The greater the proportion of go relative to no-go trials, the greater the response conflict. The typical stop-signal task is a visual choice reaction time task where trials present one of two stimuli, and each corresponds to one of two response buttons. On the go trials, participants are instructed to press the correct response button corresponding to the presented stimulus. On the stop

trials (corresponding to the no-go trials in the go/no-go paradigm), an auditory tone called a stop signal is presented at varying times after presentation of the stimulus. The stop signal indicates that the participant is supposed to inhibit responding on the trial. Thus, the stop-signal task is similar to a go/no-go task, as both require participants to inhibit a prepotent response. Unlike the go/no-go task, however, the stop-signal task presents a lag between the onset of the go stimulus and the onset of the signal to inhibit responding.

1.3.2.1.2 Sustained Attention. Sustained attention is a higher-order attention process requiring discrimination, selectivity, and concentration. Most of the tasks examining higher-order attention processes such as executive attention involve response conflict, and include tasks such as the attention network task and flanker, Stroop, and spatial conflict tasks. There is evidence that neural functioning on executive attention tasks is related to self-regulation (e.g., Buss, Dennis, Brooker, & Sippel, 2011). It is likely, however, that sustained attention and other higher-order attention processes depend, in part, on more basic attentional processes such as attentional and perceptual discrimination. In order to be selective where one attends, one must first discriminate between relevant and irrelevant stimuli. One of the most common auditory discrimination paradigms in neurophysiological research is the oddball task. In oddball paradigms, two stimuli are presented so that one is frequent and the other is infrequent. The task examines the extent to which participants neurally discriminate between the two stimuli. In some variants of the task, a behavioral response is required to the infrequent stimulus, which requires sustained attention on the part of the participant to attend to the relevant stimulus and filter out the irrelevant stimulus. The paradigm reflects sustained attention particularly among young children for whom the task is more effortful. Thus, although the oddball paradigm is not a sustained attention task

per se, it does behaviorally require sustained attention processes to behaviorally distinguish between the two stimuli and respond to the infrequent stimulus. Because we use an oddball task in the present study to examine neural attentional processes related to sustained attention, the review of the neurophysiology of sustained attention focuses on the oddball task. Nevertheless, the relation of the oddball task to the construct of sustained attention is somewhat speculative, and is one of the hypotheses tested in the present study. Whether or not the oddball task measures sustained attention, however, children with externalizing problems characterized by sustained attention deficits show attentional processing deficits tapped by neural functioning in the oddball task, as we review later.

1.3.2.2 Neurophysiology of Inhibitory Control

1.3.2.2.1 fMRI and TMS. A review and meta-analysis by Swick, Ashley, and Turken (2011) of functional magnetic resonance imaging (fMRI) studies using response inhibition tasks among adults reported that the go/no-go and stop-signal tasks recruit different, yet overlapping brain regions, suggesting that various response inhibition tasks may reflect different task demands and cognitive constructs. They observed that most prior studies of go/no-go tasks found activation in the PFC (inferior frontal gyrus, ventrolateral PFC, dorsolateral PFC), anterior cingulate cortex (ACC), and pre-supplementary motor area (pre-SMA). On the other hand, they noted that studies of stop-signal tasks typically found activation in the PFC (inferior frontal gyrus, IFG), pre-SMA, striatum, and subthalamic nucleus. In their meta-analysis, although Swick and colleagues found that the response inhibition tasks recruited some common brain regions, including the right anterior insula and the pre-SMA, they also noted that the tasks recruited some distinct regions, as well. They

found that the stop-signal task tended to activate the cingulate-opercular network associated with salience processing, whereas the go/no-go task typically activated the frontal-parietal network associated with executive control (Seeley et al., 2007). Other authors have argued that the frontal-parietal network is also important for response inhibition in children (e.g., Ciesielski, Harris, & Cofer, 2004). Based on their findings, Swick and colleagues argued that the many differences and inconsistencies in the activation regions of the two tasks suggest that there is a large distributed network for response inhibition. Part of the difficulty with localizing brain regions of activation in response to task activity may deal with the fact that behavioral tasks are not pure measures of constructs, but rather involve and depend on many different cognitive and attentional processes (Fletcher, 1996). Consistent with the interpretation that the task activation may involve non-inhibitory processes, in their review and meta-analysis of fMRI studies using go/no-go tasks among adults, Criaud and Boulinguez (2013) argued that most of the task activation (including pre-SMA) is related to attentional and working memory processes rather than inhibition.

Several fMRI go/no-go studies have examined response inhibition in children. In a study of 7–12-year-olds, children recruited the PFC (IFG, medial frontal gyrus, and orbitofrontal cortex) and ACC more in the inhibitory (no-go) compared to non-inhibitory (go) trials (Casey, Trainor, et al., 1997). Moreover, greater activation in the ACC was associated with worse response inhibition (more failed inhibition trials), whereas greater volume of activation in the orbitofrontal cortex (OFC) was associated with better response inhibition. Finally, children recruited a larger volume of activation on no-go trials than did adults, particularly in the dorsolateral PFC (DLPFC), suggesting that neural processing underlying response inhibition becomes more efficient and focalized with development.

In another fMRI study that compared 8–12-year-olds' activation in no-go compared to neutral trials in a go/no-go task, children activated regions in the right IFG and medial frontal gyrus (MFG; Bunge, Dudukovic, Thomason, Vaidya, & Gabrieli, 2002). Moreover, regions including the MFG and more posterior regions were associated with better response inhibition on the no-go trials, whereas worse response inhibition was associated with greater activation in other areas of the DLPFC and left ventrolateral PFC (VLPFC).

A go/no-go study using fMRI with children ages 6 to 10 and adults found that both children and adults activated the IFG, right DLPFC, and right parietal lobe more in no-go compared to go trials (Durston, Thomas, Yang, Uluğ, Zimmerman, & Casey, 2002). Moreover, better performance on the task (collapsing across age and trial type) was associated with greater activation in the IFG, left caudate nucleus, and left ACC. A two-session longitudinal go/no-go study (sessions were approximately two years apart) with 7–12-year-old children by Durston, Davidson, Tottenham, Galvan, et al. (2004) found that there was a decrease with age in activation of regions uncorrelated with response inhibition (see Casey, Tottenham, Liston, & Durston, 2005), whereas there were increases with age in activation of the left IFG, which has been associated with better performance on response inhibition tasks in prior studies (Casey, Trainor, et al., 1997; Durston, Thomas, et al., 2002). The authors suggested that the findings reflect the recruitment of less diffuse and more relevant, efficient, and focal processing associated with ventral PFC (IFG) regions critical for the development of response inhibition.

An fMRI go/no-go study in 8–20-year-olds found that participants activated the IFG, MFG, right superior frontal gyrus (SFG), right OFC, right insula, and right ACC more in the no-go compared to the go trials (Tamm, Menon, & Reiss, 2002). Older participants

showed more focal activity in the left IFG, left insula, and left OFC, whereas they showed less activity in the left SFG, left MFG, and left ACC, suggesting more diffuse prefrontal activation in younger participants. In a stop-signal study comparing 9–15-year-old ADHD children to controls, typically-developing children activated the right IFG on successful stop trials and showed greater activation in the ACC and left VLPFC on unsuccessful compared to successful stop trials (Pliszka, Glahn, et al., 2006).

Studies in adults have examined the neural basis of inhibitory control using experimental manipulation. Findings from a study using transcranial magnetic stimulation (TMS) with adults provide additional support for the importance of the right IFG for response inhibition (Chambers et al., 2006). Following deactivation of the right IFG, participants were impaired in the ability to inhibit an initiated action, but not the ability to execute responses, in a stop-signal paradigm. Interestingly, another study found that direct electrical stimulation of the right IFG resulted in more successful inhibition on the stop trials in a stop-signal task (Wessel, Conner, Aron, & Tandon, 2013). On the other hand, another TMS study with adults found that deactivation of the left (but not right) lateral PFC resulted in preference for immediate compared to larger, delayed rewards (Figner et al., 2010), which supports findings from lesion studies that the left PFC is important for executive functioning (Barbey, Colom, Solomon, Krueger, Forbes, & Grafman, 2012). Another study found that inhibitory control training in a stop-signal task resulted in IFG activation increases during inhibition preparation and decreases during inhibition implementation (Berkman, Kahn, & Merchant, 2014).

Additional support for the causal role of ACC functioning in self-regulation comes from

a study in adults that found that electrically stimulating the ACC using intracranial implantation resulted in a greater self-reported will to persevere in the face of challenges (Parvizi, Rangarajan, Shirer, Desai, & Greicius, 2013). An MRI study found that ACC sulcal pattern, which is a stable brain characteristic determined in utero, predicted inhibitory control on Stroop tasks at ages 5 and 9 years (Borst et al., 2014). Children with asymmetrical ACC sulcal patterns (i.e., a paracingulate sulcus in the left but not right hemisphere or vice versa) showed more efficient inhibitory control than did children with symmetrical ACC sulcal patterns (i.e., presence or absence of paracingulate sulcus in both hemispheres). Thus, MRI, fMRI, TMS, and electrical stimulation studies support the importance of the PFC and ACC for response inhibition and broader self-regulation phenotypes.

In summary, fMRI studies of response inhibition tasks in children have shown replication of greater activation in the IFG (Bunge et al., 2002; Casey, Trainor, et al., 1997; Durston, Davidson, Tottenham, Galvan, et al., 2004; Durston, Thomas, et al., 2002; Pliszka, Glahn, et al., 2006; Tamm et al., 2002), OFC (Casey, Trainor, et al., 1997; Tamm et al., 2002), and ACC (Casey, Trainor, et al., 1997; Tamm et al., 2002) in no-go (or stop) compared to go trials, which is similar to the regions of activation in studies of adults (Swick et al., 2011). Thus, the regions activated in trials requiring response inhibition include the ventral PFC (IFG and OFC), dorsal PFC (MFG), and ACC. Moreover, the ventral and dorsolateral portions of the PFC—more specifically, the left IFG (Durston, Thomas, et al., 2002; but see Pliszka, Glahn, et al., 2006), MFG (Bunge et al., 2002), and OFC (Casey, Trainor, et al., 1997)—and possibly the ACC (Durston, Thomas, et al., 2002; but see Casey, Trainor, et al., 1997; Pliszka, Glahn, et al., 2006) appear to be related to successful response inhibition in children. In addition, developmental changes suggest that with age, neural

activation recruits fewer areas unrelated to response inhibition, whereas activity becomes more focal among areas important for response inhibition (Casey, Tottenham, et al., 2005). These developmental changes appear to reflect a general shift from more diffuse to more efficient, focal processing, which may reflect more effort required by children than adults to perform response inhibition tasks (Dimoska, Johnstone, Chiswick, Barry, & Clarke, 2007). Thus, with development, response inhibition may recruit a more efficient and focalized, yet distributed network involving the ventral and dorsolateral regions of the PFC in addition to the ACC that are important for response inhibition.

1.3.2.2.2 No-go N2 ERP. In go/no-go and stop-signal paradigms, the N2 (or N200) is considered to reflect response inhibition (Nakata, Inui, Wasaka, Tamura, et al., 2006; J. L. Smith, Johnstone, & Barry, 2008) or conflict monitoring (Nieuwenhuis, Yeung, van den Wildenberg, & Ridderinkhof, 2003; Rueda, Posner, Rothbart, & Davis-Stober, 2004; van Veen & Carter, 2002a; Yeung, Botvinick, & Cohen, 2004). The N2 typically occurs approximately 200–400 ms (for adults) after the onset of the inhibitory (no-go) stimulus, yet before the behavioral response. The N2 occurs over frontocentral electrodes, and has been localized to several neural generators, most commonly the ACC in both children (Jonkman, Sniedt, & Kemner, 2007; Lamm, Zelazo, & Lewis, 2006; Stieben, Lewis, Granic, Zelazo, Segalowitz, & Pepler, 2007) and adults (Bekker, Kenemans, & Verbaten, 2005; Bokura, Yamaguchi, & Kobayashi, 2001; Jonkman, Sniedt, et al., 2007; Ladouceur, Dahl, & Carter, 2007; Mathalon, Whitfield, & Ford, 2003; van Veen & Carter, 2002b)—particularly the dorsal ACC (Stieben et al., 2007; van Veen & Carter, 2002b)—but also the right OFC (Bokura et al., 2001; Lamm et al., 2006) and the lateral PFC, including the VLPFC (particularly the IFG; Kiefer, Marzinzik, Weisbrod, Scherg, & Spitzer, 1998; Lavric, Pizzagalli, & Forstmeier,

2004; Nakata, Inui, Wasaka, Akatsuka, & Kakigi, 2005) and DLPFC (Lavric et al., 2004; Mathalon et al., 2003).

The N2 has been found to be larger on trials requiring inhibition of a prepotent response than on trials that do not require inhibition, leading some researchers to argue that the no-go (or stop) N2 may index response inhibition (Band & van Boxtel, 1999). Alternatively, other researchers have suggested that the N2 reflects conflict monitoring and not inhibition (e.g., Nieuwenhuis et al., 2003). In the following review, we review studies that examined whether the N2 reflects response inhibition.

There is some evidence that the N2 in tasks requiring inhibition may index response inhibition (for a review, see Band & van Boxtel, 1999). First, the N2 has been associated with behavioral performance during inhibition trials in both go/no-go and stop-signal tasks (e.g., van Boxtel, van der Molen, Jennings, & Brunia, 2001). Moreover, the N2 amplitude is influenced by the degree of inhibition required to overcome the prepotent response, as shown by larger N2 amplitudes on trials with greater inhibition difficulty by manipulating the time allowed to respond (Jodo & Kayama, 1992). Interestingly, a no-go ERP potential has been identified in monkeys that is considered equivalent to the no-go N2 (Band & van Boxtel, 1999), whose neural source in the PFC, when stimulated, results in successfully inhibited responses (Sasaki, Gemba, & Tsujimoto, 1989).

In addition, the N2 has also been associated with success or failure of inhibition. Larger no-go N2 amplitudes, larger N2 amplitude effects (no-go N2 amplitude minus go N2 amplitude), and shorter N2 latencies are typically associated with better response inhibition. Larger N2 amplitudes may reflect a greater recruitment of neural resources for response

inhibition, which may enable better inhibitory control. Shorter N2 latencies may reflect faster inhibitory processing. In the horse-race model, there are two ongoing and competing processes during action evaluation and execution: (1) an execution process and (2) an inhibitory process (Logan & Cowan, 1984). The process that completes first "wins the race," and determines whether a behavioral response occurs. Shorter N2 latencies may reflect faster inhibitory processing and, therefore, a greater likelihood of successful response inhibition. Consistent with these models of the N2 as reflecting response inhibition, previous studies have found larger N2 amplitudes and shorter N2 latencies in better response inhibition performance.

In a study with 4–5-year-old children, larger N2 amplitudes were associated with faster response inhibition on a go/no-go task (Lahat, Todd, Mahy, Lau, & Zelazo, 2010). In a go/no-go study with 7- and 9-year-olds, larger N2 amplitudes were associated with better inhibition performance on the no-go trials for the 7-year-olds, although the authors found no association among the 9-year-olds (Cragg et al., 2009). In addition, in a go/no-go study with adults, larger no-go N2 amplitudes and shorter no-go N2 latencies were associated with better response inhibition in the task (Falkenstein, Hoormann, & Hohnsbein, 1999). In a go/no-go study with 5-year-old children, Chevalier, Kelsey, Wiebe, and Espy (2014) observed a frontal negativity around 350–650 ms, with shorter latencies associated with better inhibition performance.

In summary, there is evidence that the no-go N2 or N2 effect may reflect response inhibition, as measured by go/no-go tasks. In particular, larger no-go N2 amplitudes and N2 amplitude effects along with shorter N2 latencies on go/no-go tasks are typically associated with better response inhibition. Consistent with this interpretation of the N2 as reflecting

response inhibition, behavior problems characterized by deficits in response inhibition such as ADHD and externalizing problems typically show smaller N2 amplitudes and longer N2 latencies, as we review later.

Similar to the go/no-go task, the stop-signal task elicits larger N2 amplitudes to stop (equivalent to no-go trials) than to go trials (Kok, Ramautar, Ruiter, Band, & Ridderinkhof, 2004). As such, studies have investigated the relation of the stop N2 to response inhibition. For example, in a stop-signal study with adults, the stop N2 was larger for successful inhibition trials compared to failed inhibition trials (Schmajuk, Liotti, Busse, & Woldorff, 2006). Moreover, adults with more efficient response inhibition on the stop-signal task (i.e., faster reaction times) have shown larger stop N2 amplitudes (van Boxtel et al., 2001). In contrast, other stop-signal studies with adults have found that stop N2 amplitudes were larger for failed than for successful inhibition trials (Dimoska, Johnstone, & Barry, 2006; Kok et al., 2004; Ramautar, Kok, & Ridderinkhof, 2004; Ramautar, Kok, & Ridderinkhof, 2006). In addition, Ramautar, Kok, and Ridderinkhof (2004, 2006) found that stop N2 latencies were longer for failed than for successful inhibition trials among adults. In summary, there are inconsistencies in the direction of association between the stop N2 and response inhibition performance. Whereas some stop-signal studies support the typical finding in go/no-go studies that larger N2 amplitudes and shorter N2 latencies are associated with better response inhibition, other stop-signal studies suggest that smaller N2 amplitudes may reflect better response inhibition. Differences between findings from go/no-go and stop-signal paradigms may reflect that the tasks may induce different types of conflict, recruit different neural resources, and therefore may reflect different cognitive constructs (Swick et al., 2011). Nonetheless, future studies should explore the association to understand the role of

the stop N2 in inhibitory processing.

In addition to response inhibition, the N2 has been examined in relation to other self-regulatory phenotypes, as well. Grabell (2014) found that smaller no-go N2 amplitudes were associated with poorer emotional dysregulation in 3 1/2–5-year-old children, as reported by parents. A study by Wiersema and Roeyers (2009) found that larger no-go N2 amplitudes were associated with better effortful control in 8–13-year-old children, as measured by self-reports of the executive attention ability to shift attention. A study by Espinet, Anderson, and Zelazo (2012) examined the relations of the N2 amplitude to executive functioning, another form of behavioral regulation requiring response inhibition, among 35–54-month-old children in the context of the Dimensional Change Card Sort task that requires sorting cards by one dimension and then sorting by a second dimension. The authors found that children with better executive functioning who passed the task had *smaller* N2 amplitudes than did children who did not. There are several possible reasons for the discrepancy in findings in comparison to other findings of children with better response inhibition showing larger N2 amplitudes. First, the study involved a different type of outcome measure that involves relatively more cognitive flexibility than response inhibition. Second, the relation between the N2 and social functioning may depend on the range of behavior problems in the sample, as described later. Thus, the findings in a normative sample may differ from those of a clinical sample.

In summary, although there are some inconsistencies in the directions of the association between the N2 and response inhibition, findings typically suggest that larger N2 amplitudes and shorter N2 latencies are associated with better response inhibition. Future studies should examine the role of the N2 in inhibitory processing versus conflict monitoring to

specify the functional role of the no-go and stop N2 in order to clarify the meaning of its relation to behavioral phenotypes.

1.3.2.3 Neurophysiology of Sustained Attention

Because of the hypothesis of the present study that the oddball paradigm with a behavioral response to an infrequent stimulus recruits key components of the sustained attention response, we limit the review here to studies examining attentional processes with oddball paradigms (as opposed to other attention tasks such as the attention network task, flanker, Stroop, and spatial conflict tasks).

1.3.2.3.1 fMRI. We identified three fMRI studies examining oddball paradigms in children with ADHD. Rubia, Smith, Brammer, and Taylor (2007) compared 17 boys with ADHD to 18 typically-developing boys 9–17 years of age. On the frequent compared to infrequent trials, boys with ADHD showed less activation in the ACC and DLPFC compared to typically-developing boys. On the infrequent compared to frequent trials, boys with ADHD showed less activation than typically-developing boys in temporal lobes, the basal ganglia, and posterior cingulate. In a study by Stevens, Pearlson, and Kiehl (2007), 23 boys with ADHD were compared to 23 typically-developing boys 11–18 years of age. To the infrequent stimuli, boys with ADHD showed less activation than typically-developing boys in the PFC (left middle frontal gyrus) and temporal lobes (anterior portion of right superior temporal gyrus and middle portion of right middle temporal gyrus). In a study by Tamm, Menon, and Reiss (2006), 14 boys with ADHD were compared to 12 typically-developing boys 14–18 years of age. To infrequent compared to frequent trials, boys with

ADHD showed less activation than typically-developing boys in the parietal and cingulate cortex, among other areas. Moreover, among typically-developing boys (but not boys with ADHD), greater degree of activation in the left parietal cortex was associated with fewer commission errors on the infrequent trials. In summary, infrequent trials with a behavioral response in oddball paradigms may involve an attentional network involving the PFC, cingulate cortex, and temporo-parietal regions that collectively support sustained attention.

1.3.2.3.2 Oddball P3 ERP. The P3 (or P300) is considered to reflect attentional processes in oddball paradigms (Key, Dove, & Maguire, 2005). It is somewhat speculative that the P3 may be related to sustained attention processes. During oddball tasks that require a behavioral response to the infrequent stimulus, a P3 (P3b) component occurs 300–500 ms (for adults) after the onset of the target (infrequent) stimulus (Soltani & Knight, 2000). This P3 component has a parietal scalp distribution. During passive oddball tasks with no behavioral response, in contrast, a P3 (P3a) component occurs 60–80 ms earlier than does the P3b after the onset of the infrequent stimulus, with a frontocentral scalp distribution (Soltani & Knight, 2000). The P3b is considered to reflect various attentional processes, such as degree of attentional engagement (Key et al., 2005), which is conceptually related to focused, sustained attention. The P3a is considered to reflect involuntary attentional processes, and may thus be less relevant to sustained attention processes than the P3b. Some of the evidence that the P3b may be related to sustained attention comes from evidence that there are differences in the P3 in children with ADHD, a disorder whose key deficits include sustained attention deficits that arise from deficient inhibitory control (Barkley, 1997). Studies have often shown that children with ADHD have smaller P3 amplitudes and longer P3 latencies compared to controls, as we review later.

Note that a P3 waveform is also observed in many go/no-go tasks in older children and adults, but the no-go P3 is interpreted as reflecting response inhibition (Bokura et al., 2001; Donkers & van Boxtel, 2004; Kiefer et al., 1998) unlike the oddball P3, which is considered related to attentional processing (Key et al., 2005). We have decided to focus on the no-go N2 rather than the no-go P3 in the present studies because we did not observe a no-go P3 ERP in our go/no-go tasks with toddlers. Our inability to detect a no-go P3 in 2 1/2 to 3 1/2 year-old children may reflect the finding that the no-go P3 amplitude increases with age and is relatively small in children (Hämmerer, Li, Müller, & Lindenberger, 2010).

1.3.2.4 Brain Structure and Externalizing Problems

Many studies have examined the neural structure in ADHD and other externalizing problems. A common finding in these studies is differences, typically smaller volume or thinner cortex, in the PFC and ACC (for a review and meta-analysis of studies in adults, see Yang & Raine, 2009), but other regions are involved, as well (e.g., amygdala; Pardini, Erickson, Loeber, and Raine, 2014; temporal regions; Michalska, Decety, Zeffiro, and Lahey, 2015). Smaller PFC gray matter volume has been associated with antisocial personality disorder in adults (Raine, Lencz, Bihrle, LaCasse, & Colletti, 2000). A study examining the brain structure underlying conduct disorder (CD) symptoms in 22, 14–20-year-old adolescent girls using MRI found that aggressive CD symptoms were associated with smaller DLPFC volume (Fairchild, Hagan, Walsh, Passamonti, Calder, & Goodyer, 2013). A study comparing 23 boys from ages 12 to 17 with CD and 23 typically-developing boys found a smaller OFC in the boys with CD compared to the typically-developing boys (Huebner et al., 2008).

Regarding the brain structure underlying ADHD, a common finding is that children with ADHD have a thinner cortex than do typically-developing children (Narr et al., 2009). An MRI study found that 32 children with ADHD had a thinner ACC than did 15 typically-developing children from ages 9–15 (Bledsoe, Semrud-Clikeman, & Pliszka, 2013). Moreover, over one-third of the variance in parents' ratings of ADHD symptoms was explained by ACC thickness. The authors interpreted the thinner ACC in ADHD compared to that found in typically-developing children as reflecting a compromised anterior attention network involved in error detection, impulsivity, and inhibitory control. A prospective study found that a shorter corpus callosum in 784, 6-week-old infants was associated with poorer executive functioning and inhibition at 4 years of age (Ghassabian et al., 2013). The length of the corpus callosum did not predict later ADHD symptoms, however. An MRI study found that, compared to 22 community-reared children, 58 children reared in institutions showed less cortical thickness in many brain regions at 8–10 years of age, including the PFC (McLaughlin, Sheridan, Winter, Fox, Zeanah, & Nelson, 2014). The smaller cortical thickness mediated the effects of institutionalization on teacher-reported ADHD symptoms.

Several longitudinal studies have examined the neurophysiological development of ADHD (Shaw, Eckstrand, et al., 2007; Shaw, Lerch, et al., 2006; Shaw, Malek, Watson, Greenstein, Rossi, & Sharp, 2013). One study by Shaw and colleagues (Shaw, Lerch, et al., 2006) compared the cortical thickness of 163 children with ADHD and 166 typically-developing children using MRI. Four MRI assessments were conducted at the following ages: (1) 7–13 years old, (2) 9–16, (3) 11–18, and (4) 13–22. The study found that, at intake, children with ADHD had a thinner cortex than did controls, particularly in the PFC, cingulate cortex, and precentral regions. Moreover, children with ADHD at intake who showed worse clinical

outcomes during follow-up assessments had thinner cortices in similar prefrontal regions compared to children with ADHD at intake who showed better outcomes in later assessments, as measured by the Children's Global Assessment Scale. The authors interpreted the thinner PFC and ACC as possibly reflecting the attention or response inhibition deficits found in ADHD. The authors interpreted the thinner precentral gyrus in ADHD as possibly reflecting motor hyperactivity or deficient response inhibition. The authors found no differences, however, in the growth rate of cortical thickness over time between children with ADHD and controls, with the exception of regions of the parietal cortex (whose thickness eventually converged with that of controls).

A subsequent study by Shaw's research team compared the growth trajectories of cortical thickness among 223 children with ADHD compared to 223 typically-developing children (Shaw, Eckstrand, et al., 2007). The authors found that although trajectories of the rates of cortical thickening were similar between ADHD and typically-developing children, the children with ADHD showed delayed cortical thickening compared to typically-developing children. Typically-developing children reached peak cortical thickness by 7 1/2 years of age, on average, whereas children with ADHD reached peak cortical thickness by 10 1/2 years, on average. The greatest delays in cortical thickening were observed in the middle PFC, where ADHD children showed delays of approximately 5 years relative to typically-developing children in terms of cortical thickness. The finding of a lag in brain development in ADHD has been replicated using connectomic analyses of brain networks (Sripada, Kessler, & Angstadt, 2014).

A third study by Shaw's research group examined the trajectories of cortical thickening from childhood into adulthood among 92 participants diagnosed with ADHD as children in

relation to ADHD symptoms in adulthood (Shaw, Malek, et al., 2013). The study observed that cortical thinning over time in the medial and dorsolateral PFC was associated with greater inattentive, but not hyperactive/impulsive, ADHD symptoms in adulthood.

In addition to longitudinal studies of the brain structure in ADHD, another longitudinal study examined the brain structure in general externalizing problems (Ameis et al., 2014). The study followed 297 children at ages 6–18 years using MRI at 2-year intervals for up to 3 assessments (517 total scans). Despite the study's longitudinal design and analytical approach that took into account the dependence of repeated measures data (hierarchical linear modeling), the study only examined whether brain structure was associated with the level (i.e., intercept) of externalizing problems and not the development of externalizing problems over time. Externalizing problems were associated with a thinner cortex in the left OFC and right cingulate cortex. In addition, children with fewer externalizing problems showed a positive association between OFC and amygdala thickness, whereas children with more externalizing problems showed no association between OFC and amygdala thickness. This finding is consistent with findings of reduced functional coupling of the OFC and amygdala in children with ODD/CD and psychopathic traits (Marsh et al., 2013; but see Sarkar et al., 2013).

Another longitudinal study examined change in ACC thickness, self-regulation, and externalizing problems from ages 12–16 years in 92 adolescents who were selected based on their representing a full range of risk on temperamental negative emotionality and self-regulation (Vijayakumar, Whittle, Dennison, Yücel, Simmons, & Allen, 2014). Self-regulation was measured by self reports of their temperamental effortful control. Externalizing problems—aggression and risk taking—were also measured by self report. Adolescence

is a period of normative pruning of inefficient synaptic connections, and increases in white matter (via myelination) that result in cortical thinning of gray matter. The authors found that less cortical thinning in the left ACC from age 12 to 16 was associated with the development of self-regulation deficits and externalizing problems. Moreover, changes in self-regulation mediated the effect of ACC thinning on changes in externalizing problems, suggesting that self-regulation may be an underlying, intermediate phenotype of externalizing problems. These findings are consistent with findings from a study of 5–10-year-old children, in which children with thinner cortex in the PFC (right IFG) and ACC had better inhibitory control performance on a Simon task (Kharitonova, Martin, Gabrieli, & Sheridan, 2013). In addition, the association between age and inhibitory control was mediated by PFC and ACC thickness. Thus, the development of inhibitory control and externalizing problems may be explained by the extent of synaptic pruning and cortical gray-matter thinning in the PFC and ACC.

In sum, compared to typically-developing children, children with ADHD and other externalizing problems show a smaller or thinner cortex in brain regions such as the PFC and ACC that are associated with sustained attention, response inhibition, and higher-order cognitive control processes. Interestingly, the thinner cortex in ADHD compared to typically-developing children appears to result from a *delay* in cortical development rather than a qualitatively distinct trajectory of brain development. In other words, children with ADHD and typically-developing children show similar rates of cortical development when cortical thickening begins, but children with ADHD may have a delayed onset of cortical thickening. Moreover, less cortical gray-matter thinning in the PFC and ACC during adolescence, a normative period of synaptic pruning, may be associated with self-regulation

deficits and externalizing problems. These neural differences (delayed onset of cortical thickening and less cortical thinning in the PFC and ACC during synaptic pruning) may underlie the symptoms present in ADHD and other disorders of disinhibition. The effects of the neural differences in ADHD and externalizing problems may be explained through their effects on self-regulation.

1.3.2.5 Brain Functioning and Externalizing Problems

In terms of function, externalizing problems and related phenotypes in children are often associated with less activation in the PFC and ACC during inhibitory and attention tasks according to studies using fMRI and TMS (Bunge et al., 2002; Bush, Frazier, et al., 1999; Cao et al., 2008; Casey, Trainor, et al., 1997; Castellanos-Ryan et al., 2014; Chambers et al., 2006; Durston, Davidson, Tottenham, Galvan, et al., 2004; Durston, Thomas, et al., 2002; Pliszka, Glahn, et al., 2006; Tamm et al., 2002). Findings of less activation in the PFC and ACC in externalizing problems among children are consistent with findings from a meta-analysis of studies in adults (Yang & Raine, 2009). ADHD is also characterized by hypoactivation in the ACC and frontal lobes, in addition to other regions (for a review, see Weyandt, Swentosky, & Gudmundsdottir, 2013). In addition to hypoactivation of the PFC and ACC, ADHD may also be characterized by less connectivity between the PFC and ACC. Reduced functional coupling between the PFC and cingulate cortex has been observed among ADHD patients whose symptoms persisted into adulthood compared to ADHD patients whose symptoms remitted (Mattfeld et al., 2014). Moreover, *DRD4*, the gene most strongly linked to ADHD, has shown links to PFC and ACC functioning. Carriers of the risk *DRD4* 7-repeat allele showed less activation in and coupling between the PFC and

ACC (Gilsbach et al., 2012). Perhaps the findings of hypoactivation of the PFC and ACC provides a clue to how externalizing problems develop. Neural functioning in externalizing problems is often studied with ERPs—direct measures of neural activity to stimuli generated from postsynaptic potentials that are detected on the scalp from EEG. ERPs with neural sources in the PFC and ACC, including the N2 (the second negativity in the waveform) and P3 (the third positivity) have functional interpretations similar to the cognitive deficits in externalizing disorders, including reduced inhibitory control and attention deficits (Dias, Foxe, & Javitt, 2003; Lavric et al., 2004; Stieben et al., 2007).

Neural processes typically become more efficient and focalized to task-relevant regions with development (Durston, Davidson, Tottenham, Spicer, et al., 2006). However, smaller N2 and P3 amplitudes and longer latencies reflect diffuse (i.e., less focalized activation) and inefficient neural activity in relevant regions of the PFC and ACC. Thus, smaller N2 and P3 amplitudes and longer latencies likely reflect specific deficits in inhibitory control (N2) and sustained attention (P3). It is hypothesized that these deficits result in less capable and efficient self-regulation—an intermediate phenotype of externalizing behavior (Calkins & Howse, 2004; Doyle et al., 2005; Gagne et al., 2011; Hardaway, Wilson, Shaw, & Dishion, 2012; Olson, Sameroff, Kerr, Lopez, & Wellman, 2005; Patrick, Venables, et al., 2013; Slaats-Willemse, Swaab-Barneveld, Sonneville, van der Meulen, & Buitelaar, 2003; Sulik, Blair, Mills-Koonce, Berry, Greenberg, & The Family Life Project Investigators, in press; Vijayakumar et al., 2014; Young et al., 2009).

Generally (but see Senderecka, Grabowska, Szewczyk, Gerc, & Chmylak, 2012; J. L. Smith, Johnstone, & Barry, 2004), smaller N2 and P3 amplitudes and longer N2 and P3 latencies are often associated with externalizing phenotypes (poor response inhibition) and

psychopathology (ADHD, ODD, conduct disorder, and alcohol and substance abuse), in oddball, go/no-go, and stop-signal paradigms among older children and adults (Albrecht, Banaschewski, Brandeis, Heinrich, & Rothenberger, 2005; Barry, Johnstone, et al., 2003; Bekker, Overtoom, Kooij, Buitelaar, Verbaten, & Kenemans, 2005; Cragg et al., 2009; de Jong, Coles, Logan, & Gratton, 1990; Dimoska, Johnstone, Barry, & Clarke, 2003; Fallgatter et al., 2004; Grabell, 2014; Jetha, Segalowitz, Gatze-Kopp, & Ly, 2010; Johnstone, Barry, Markovska, et al., 2009; Kok et al., 2004; Liotti, Pliszka, Perez, Kothmann, & Woldorff, 2005; Overtoom, Kenemans, et al., 2002; Overtoom, Verbaten, et al., 1998; Pliszka, Liotti, & Woldorff, 2000; Ramautar et al., 2004, 2006; Schmajuk et al., 2006; Senderecka, Grabowska, Gerc, Szewczyk, & Chmylak, 2012; van Boxtel et al., 2001; van der Schoot, Licht, Horsley, & Sergeant, 2002; Wild-Wall, Oades, Schmidt-Wessels, Christiansen, & Falkenstein, 2009; Woltering, Granic, Lamm, & Lewis, 2011). In addition to the no-go N2 and oddball P3, ERP studies of externalizing problems have also examined the error-related negativity (ERN; Hall, Bernat, & Patrick, 2007), mismatch negativity (MMN; Hung, Ahveninen, & Cheng, 2013), and no-go P3 (Woltering et al., 2011). The present review focuses on the no-go N2 and the oddball P3 because of their inclusion in the present studies.

We did not examine the ERN for three primary reasons: (1) we are most interested in neural processing during early stimulus processing that might predict the behavioral response (whereas the ERN occurs *after* the behavioral response), (2) the ERN is not reliably observed in children (Davies, Segalowitz, & Gavin, 2004; Ladouceur, Dahl, & Carter, 2004), and (3) when the ERN is observed in young children, it does not reflect the degree of error processing (Torpey, Hajcak, & Klein, 2009), suggesting that its meaning differs in early childhood. In addition, we did not examine the MMN in the present studies. The MMN

is similar to the P3a in that it is a fairly automatic neural process in response to novel stimuli. We opted to focus on the P3a instead of the MMN because the P3a is thought to reflect attentional processes and orienting to novelty, whereas the MMN is thought to reflect basic sensory/perceptual mechanisms in deviance detection (Hermens, Ward, Hodge, Kaur, Naismith, & Hickie, 2010).

1.3.2.5.1 No-go N2 ERP. Smaller N2 amplitudes have been observed in self-regulation deficits and externalizing problems in numerous studies of children. Grabell (2014) found smaller no-go N2 amplitudes in 3–5-year-old children who were clinically referred for externalizing behavior problems compared to typically-developing children. Grabell also found that smaller no-go N2 amplitudes were associated with more externalizing problems measured dimensionally, as reported by parents. Berger, Alyagon, Hadaya, Atzaba-Poria, and Auerbach (2013) observed a correlation between N2 amplitude and ADHD symptoms among 5-year-old boys with familial risk for ADHD in a stop-signal task. Smaller N2 amplitudes were associated with more ADHD symptoms, hyperactivity in particular. A study of 6–14-year-old children with ADHD used a continuous performance task (CPT-AX), which is a go/no-go task where the go stimulus is the letter 'X' only when directly preceded by the letter 'A', and every other letter or sequence of letters is a no-go trial (Overtoom, Verbaten, et al., 1998). The authors observed smaller N2 amplitudes among children with ADHD and ODD compared to typically-developing children. In a flanker task with no-go trials, Wild-Wall et al. (2009) observed smaller N2 amplitudes in children with ADHD than typically-developing children from ages 11–17 years. Johnstone, Barry, Markovska, et al. (2009) observed a smaller N2 amplitude effect (no-go N2 amplitude minus go N2 amplitude) among children with ADHD compared to typically-developing children from ages 8–14

years. A study of 5–6-year-old children found that among children at risk for externalizing problems, children with smaller no-go N2 amplitude effects had more severe externalizing behavior problems (Jetha et al., 2010). Albrecht et al. (2005) observed smaller stop N2 amplitudes in ADHD, ADHD+ODD, and ODD/CD compared to typically-developing children ages 8–14 years. Pliszka, Liotti, et al. (2000) observed smaller stop N2 amplitudes in children with ADHD compared to typically-developing children among 10–12-year-olds. Dimoska, Johnstone, Barry, and Clarke (2003) observed smaller stop N2 amplitudes in boys with ADHD compared to typically-developing boys from ages 7–12 years. Johnstone, Barry, and Clarke (2007) observed smaller stop N2 amplitudes in 8–14-year-old children with the predominantly inattentive subtype of ADHD compared to typically-developing children. A study using a go/no-go task found that 8–12-year-old children with externalizing problems and children with comorbid internalizing and externalizing problems recruited less dorsal ACC activity associated with the N2 than did controls (Stieben et al., 2007). Broyd et al. (2005) observed smaller no-go N2 amplitudes in 8–14-year-old boys with ADHD compared to typically-developing boys. There were no differences in no-go N2 amplitudes between boys with ADHD and controls, however, after those with ADHD received pharmacological treatment of methylphenidate. This suggests that pharmacological treatment for ADHD may work, in part, via its effects on the N2 and response inhibition.

Other studies, on the other hand, have suggested that *larger* N2 amplitudes may be a risk marker for ADHD and externalizing problems. A study by J. L. Smith, Johnstone, and Barry (2004) found that 7–12-year-old ADHD children showed larger N2 amplitude effects and shorter N2 latencies in a go/no-go task compared to controls. A go/no-go study with

8–12-year-old children found that children with externalizing problems had larger N2 amplitudes than did typically-developing children prior to treatment (Woltering et al., 2011). Following cognitive behavioral therapy and parent behavior training, however, children with externalizing problems who improved following treatment showed no N2 differences compared with controls. Improvers also showed smaller N2 amplitudes than did non-improvers post-treatment. Moreover, the improvers showed less activation than did non-improvers in a ventromedial region of the PFC suggestive of the dorsal ACC. The authors interpreted the smaller N2 amplitudes and associated decrease in activation in the (possible) dorsal ACC among the improved children with externalizing problems as reflecting increasing cortical efficiency of response inhibition, as supported by evidence that N2 amplitudes decrease with age (Broyd et al., 2005; Ciesielski et al., 2004; E. P. Davis, Bruce, Snyder, & Nelson, 2003; Hämmerer, Li, Müller, et al., 2010; Henderson, 2010; Johnstone, Dimoska, et al., 2007; Johnstone, Pleffer, Barry, Clarke, & Smith, 2005; Jonkman, 2006; Jonkman, Lansbergen, & Stauder, 2003; Jonkman, Sniedt, et al., 2007; Lamm et al., 2006; Lewis, Lamm, Segalowitz, Stieben, & Zelazo, 2006). The finding of dorsal ACC abnormalities in externalizing problems is unsurprising given the role of the dorsal ACC in cognitive control (Bush, Luu, & Posner, 2000). Possible reasons for the discrepancies in the direction of the association between N2 amplitudes and externalizing problems in prior studies are described later.

Studies have also identified longer N2 latencies in externalizing problems in studies of children. Fallgatter et al. (2004) observed longer no-go N2 latencies in children with ADHD compared to typically-developing children from ages 7–11 years. Moreover, two studies found longer N2 latencies were associated with poorer response inhibition among adults in a stop-signal task (Ramautar et al., 2004, 2006).

Interestingly, the N2 appears to be modulated by early environmental deprivation. Loman, Johnson, Westerlund, Pollak, Nelson, and Gunnar (2013) compared the N2 amplitude between 24 post-institutionalized children, 31 children adopted early from foster care, and 27 nonadopted children from ages 10–11 years. The adopted children came to the United States via international adoptions. The post-institutionalized group was adopted after 12 months of age, whereas the early adoption group was adopted between 2–7 months of age. The authors found that the post-institutionalized group had smaller no-go N2 amplitudes (possibly indicating poorer response inhibition) than did early adopted and nonadopted children.

1.3.2.5.2 Oddball P3 ERP. The oddball P3, which is sensitive to deficient attentional processing (reviewed earlier), also exhibits differences in externalizing problems and attention deficits. A robust finding is a smaller oddball P3 amplitude in individuals with externalizing problems (Gilmore, Malone, & Iacono, 2010), aggression (Bernat, Hall, Steffen, & Patrick, 2007), substance abuse (Iacono & Malone, 2011), and ADHD (Barry, Johnstone, et al., 2003) compared to healthy controls. Johnstone and Barry (1996) found smaller oddball P3 amplitudes in 6–13-year-old children with ADHD compared to typically-developing children. Jonkman, Kemner, et al. (1997) observed smaller oddball P3 amplitudes in 7–13-year-old children with ADHD compared to typically-developing children. Another study, however, found that the smaller P3 amplitude found in 10–12-year-old children with ADHD may reflect the effects of comorbid disruptive behavior problems rather than ADHD symptoms *per se* (Yoon, Iacono, Malone, Bernat, & McGue, 2008).

Given smaller P3 amplitudes are a biomarker of the genetic vulnerability to externalizing disorders (Hicks et al., 2007; Iacono & Malone, 2011), it has been argued that the P3 represents an endophenotype for externalizing psychopathology (Gilmore, Malone, & Iacono, 2010). If smaller P3 amplitudes are an endophenotype of externalizing problems, in addition to showing sensitivity as a marker of externalizing, the oddball P3 may also have specificity for externalizing problems. Another argument for the specificity of the P3 for externalizing problems is based on evidence that children with ADHD have smaller P3 amplitudes than do children with other developmental disorders such as autism and dyslexia (Kemner, Verbaten, Koelega, Camfferman, & van Engeland, 1998). Moreover, the P3 is modulated by dopaminergic neurotransmission that is involved in attention and impulsivity problems (i.e., greater dopamine D2/D3-receptor functioning is associated with larger P3 amplitudes and shorter P3 latencies; Pogarell et al., 2011), suggesting that the underlying generators of the P3 may involve mechanisms relating to externalizing phenotypes. On the other hand, (1) the dopaminergic system is fairly diffuse and projects to many brain regions (so the mechanisms may not be specific), and (2) the P3 is also associated with other problems, including depression and schizophrenia. However, the link between the P3 and externalizing problems holds controlling for depression, and it has been suggested that the P3 may reflect a state indicator of depression as opposed to a trait indicator of externalizing problems (Patrick, Bernat, Malone, Iacono, Krueger, & McGue, 2006). Moreover, the P3 mechanisms in externalizing problems may be different (i.e., more specific to the visual modality) than for schizophrenia (Patrick, Bernat, et al., 2006). Thus, the attention deficits (marked by smaller P3 amplitudes) may represent an intermediate phenotype with functional significance for multiple problems, including externalizing problems.

In summary, ERP findings tend to suggest smaller amplitudes and longer latencies of the N2 and P3 ERPs in externalizing problems. The findings between the N2/P3 ERPs and externalizing problems are not entirely consistent, however. There are cases of null associations (Spronk, Jonkman, & Kemner, 2008) and instances where larger N2 amplitudes (Senderecka, Grabowska, Szewczyk, et al., 2012; J. L. Smith et al., 2004; Woltering et al., 2011) and shorter latencies (J. L. Smith et al., 2004) are associated with externalizing-related phenotypes. In general, however, studies show that children with ADHD and externalizing problems have smaller N2/P3 amplitudes and amplitude effects, in addition to longer N2/P3 latencies than do healthy controls (for a review of ERP abnormalities in ADHD, see Barry, Johnstone, et al., 2003), suggesting that the magnitude and efficiency of cognitive processing associated with the N2 and P3 may account for some of the response inhibition and sustained attention deficits in externalizing disorders. Although reasons for the inconsistencies are unknown, differences in findings may reflect methodological differences such as the frequency of go relative to no-go trials, or they may result from sample differences reflecting the heterogeneity of ADHD and externalizing problems or comorbidities with other disorders (e.g., learning disorders).

Alternatively, part of the discrepancy in findings may result from what seem to be paradoxical findings. On the one hand, previous studies have generally shown clinical externalizing disorders are characterized by smaller amplitudes of the N2 and P3 (see Figure 1.1, Panel A). On the other hand, N2 and P3 amplitudes tend to decrease with development, suggesting an increase in processing efficiency with development. Thus, smaller N2/P3 amplitudes appear to reflect worse functioning in the case of externalizing disorders yet better functioning in the case of more advanced development. Speculatively, these findings could

reflect a nonlinear, U-shaped function of the relation of the N2/P3 to prosocial behavior where there is an optimal range for N2/P3 amplitudes (see Figure 1.1, Panel B). If too large or too small, the N2/P3 amplitudes may reflect poor behavioral adjustment. For example, consider a child with the task of inhibiting a prepotent response. If the child recruits too few inhibitory resources, the child will likely fail to inhibit the response. In contrast, if the child recruits too many inhibitory resources, the processing may be less efficient/automatic and may be more effortful, taking away important processing resources from other tasks. The U-curve hypothesis is supported by findings in rats that attention deficits can result from both hypo- and hyper-activation of the medial prefrontal cortex (Pezze, McGarrity, Mason, Fone, & Bast, 2014). If the U-curve hypothesis is true, the range of symptomatology of the study's sample would determine which part of the U-curve is reflected in the association, and therefore, whether the association appears positive or negative. For this reason, it may be important to study the relation of the N2/P3 to externalizing behavior in a community sample with a wide range of behavioral adjustment. Furthermore, if the N2/P3 has a U-shaped function in relation to behavioral adjustment, it may be important to examine a nonlinear (e.g., quadratic) association between the N2/P3 and externalizing problems. The present studies will investigate whether there is a nonlinear association between the N2/P3 and self-regulation and externalizing behavior.

1.3.2.5.3 EEG. In addition to ERP studies examining externalizing problems, there are also EEG studies examining externalizing problems and related phenotypes. Studies examining EEG correlates of externalizing problems in young children have focused on the 6–9 Hz frequency band because it is related to self-regulation and is the dominant frequency band in toddlers (Fox, Henderson, Rubin, Calkins, & Schmidt, 2001). The 6–9 Hz

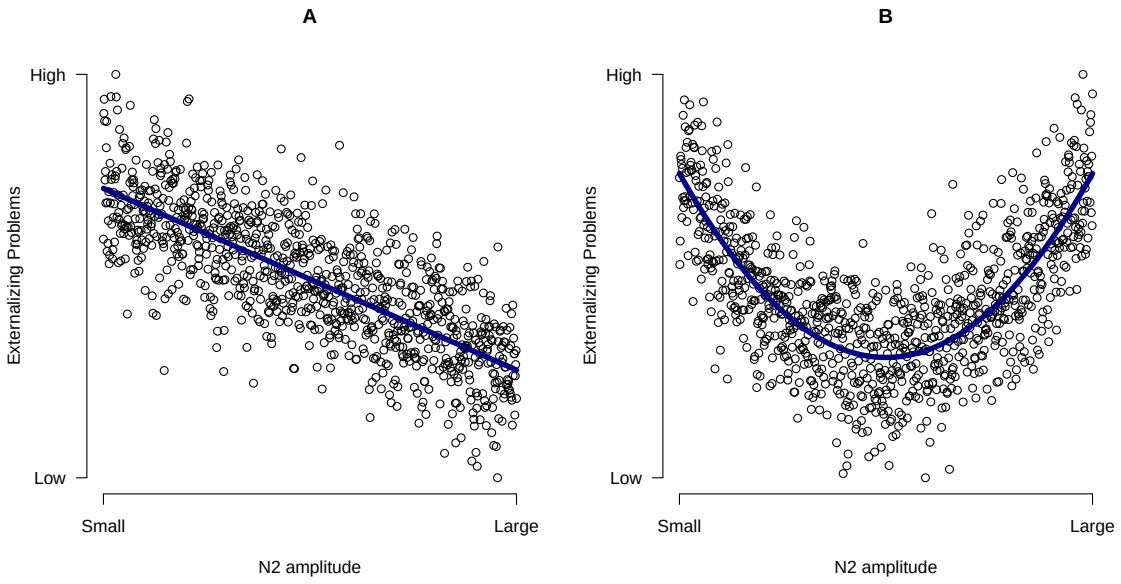

FIGURE 1.1: Panel A depicts the theoretical possibility of an inverse linear association between N2 amplitudes and externalizing problems, where smaller N2 amplitudes are associated with more externalizing problems. Panel B depicts the theoretical possibility of a nonlinear, U-shaped function of the relation of the N2 amplitudes and externalizing problems, where excessively small or large N2 amplitudes are associated with more externalizing problems.

activity in young children is known as "infant alpha" and, because of developmental shifts in frequency bands, is considered similar to a combination of theta and alpha activity in adults (Bell & Cuevas, 2012), which has been related to the selection and suppression of attention (Klimesch, 2012). Because of the relevance of infant alpha to attention and emotion regulation, it has often been examined in relation to self-regulation and externalizing behavior.

To our knowledge, C. L. Smith and Bell (2010) conducted the study that has examined neurophysiological correlates of externalizing problems at the youngest ages. The study examined hemispheric asymmetry in frontal EEG scalp electrodes in the infant alpha (6–9 Hz) frequency band in relation to mothers' reports of their child's externalizing problems.

Children with stable left frontal asymmetry in the infant alpha frequency band (i.e., greater activation at left than right frontal electrodes) at 10 and 24 months of age were reported to show more externalizing problems at 30 months of age than did other children. This finding is consistent with research showing left frontal asymmetry to be a marker of behavioral approach and right frontal asymmetry to reflect withdrawal (Coan & Allen, 2004). In contrast, a study with older (11-year-old) children found *right* frontal asymmetry in the 8–10 Hz frequency band among girls with externalizing problems, whereas they found no frontal asymmetry among boys with externalizing problems (Baving, Laucht, & Schmidt, 2003). The same authors (2000) found right frontal asymmetry in the 8–10 Hz frequency band among 4 ½- and 8-year-old girls with ODD and no frontal asymmetry among boys with ODD. Baving and colleagues (1999) also found less right frontal asymmetry in the 8–10 Hz frequency band among 4 ½- and 8-year-old boys with ADHD compared to typically-developing boys, and more right frontal asymmetry among girls with ADHD compared to typically-developing girls. Another study found that 4-year-old children with externalizing problems were more likely to have right frontal (as opposed to left frontal) asymmetry in the 6–8 Hz frequency band (Fox, Schmidt, Calkins, Rubin, & Coplan, 1996), consistent with a study of 10-year-olds (Santesso, Reker, Schmidt, & Segalowitz, 2006). Thus, despite evidence that left frontal asymmetry may reflect behavioral approach, there are inconsistent relations between frontal hemispheric asymmetry and externalizing problems. A meta-analysis found no association between left frontal hemispheric asymmetry and externalizing problems in children (Peltola, Bakermans-Kranenburg, Alink, Huffmeijer, Biro, & van Ijzendoorn, 2014).

Studies of differences in frontal infant alpha power among children with disinhibitory

deficits extend to findings of less frontal infant alpha power in poorer inhibitory control in 4 1/2-year-old children (as measured by two behavioral tasks of inhibitory control; Wolfe & Bell, 2004). Moreover, poorer inhibitory control has been associated with less baseline-to-task changes in frontal alpha power in 24–27-month-old children (as measured by a behavioral task of inhibitory control and parents' ratings of temperamental inhibitory control; Morasch & Bell, 2011). Yet there is also a study suggesting that *more* frontal alpha power in 10-month-old infants may predict poorer executive functions, as measured by lab tasks (variants of Bear/Dragon, Dimensional Change Card Sort, and yes-no tasks) at 4 years of age and parent report at 6 years of age (Kraybill & Bell, 2013). Also, a common finding in children with ADHD compared to typically-developing children is less alpha power (for a review, see Barry, Clarke, & Johnstone, 2003). A meta-analysis that combined studies of children and adults found that participants with antisocial behavior had less alpha power than controls ($g = -0.33$), whereas no significant differences were observed among participants with ADHD compared to controls (though effects were in the same direction, $g = -0.29$; Rudo-Hutt, 2015). Alpha power is inversely associated with degree of cortical activity (Fox, 1994), so findings suggest that externalizing problems are characterized by greater frontal cortical activity.

Examining a somewhat different phenotype representing behavioral dysregulation, McGough, McCracken, Cho, Castelo, Sturm, Cowen, Piacentini, and Loo (2013) examined the neural correlates of the Child Behavior Checklist–Dysregulation Profile, which reflects those children with elevated scores (T-scores of 70 or greater) on the Attention Problems, Aggression, and Anxious/Depressed subscales of the Achenbach Child Behavior Checklist. Compared to typically-developing children, children meeting criteria for the Dysregulation

Profile had less delta and more alpha power in central and parietal electrode regions during a go/no-go task. Among Dysregulation Profile children, delta and alpha power were negatively associated with parent reports on the Aggression and Anxious/Depressed subscales but not the Attention problems subscale. Delta frequency bands are considered to reflect homeostatic and motivational processes (Knyazev, 2012).

In summary, EEG findings are mixed but some suggest that left frontal hemispheric asymmetry and less infant alpha power (i.e., greater frontal cortical activity) and delta power are associated with externalizing problems. The evidence of less frontal infant alpha power may correspond to ERP findings because, in adults, the N2 and P3 ERPs are thought to reflect frontal theta activity (i.e., considered comparable to infant alpha; Harper, Malone, & Bernat, 2014; Porjesz & Rangaswamy, 2007). In addition to frontal alpha activity, other bands in frontal scalp regions such as beta (23–26 Hz; Lo et al., 2013) and gamma (31–50 Hz; Benasich, Gou, Choudhury, & Harris, 2008) may be involved in the development of inhibitory control in children, as well. Beta frequency bands may reflect processes such as motor preparation and inhibition (Lo et al., 2013), whereas gamma frequency bands are considered to reflect a wide range of processes involving attention and memory (Benasich et al., 2008).

1.3.2.5.4 Time-Frequency Analyses. In addition to examining neurophysiological correlates of externalizing problems in the time domain (ERPs) and frequency domain (EEG power analysis), studies have also examined neurophysiological correlates of externalizing problems in the time by frequency domain (time-frequency analysis). Time-frequency analyses have shown less frontal theta (4–5 Hz) and parietal delta (1–2 Hz) activity in alcoholics

compared to non-alcoholics around 300–500 ms after an oddball target stimulus (Jones et al., 2006; Porjesz, Rangaswamy, Kamarajan, Jones, Padmanabhapillai, & Begleiter, 2005). The time-frequency findings of less frontal theta and parietal delta activity in alcoholics may correspond to the findings of smaller P3 amplitudes in externalizing problems. The P3 is considered to reflect a combination of frontal theta (Harper et al., 2014; Porjesz & Rangaswamy, 2007) and parietal delta activity (Gilmore, Malone, Bernat, & Iacono, 2010), and theta activity is considered to arise, in part, from frontal brain regions and the ACC (McGough et al., 2013). Whereas P3-related theta activity is thought to represent focused attention, P3-related delta activity is considered to reflect signal matching and memory updating (Gilmore, Malone, Bernat, et al., 2010). Another study examined the neural functioning in an oddball task among 8–16-year-old boys with externalizing problems (Gilmore, Malone, Bernat, et al., 2010). The study found less parietal delta (0–3 Hz) activity during the timing of the P3 in a variety of externalizing problems (CD, ADHD, ODD, nicotine dependence, alcohol abuse/dependence, or illicit drug abuse/dependence).

In summary, previous studies have shown candidate neural mechanisms in the development of externalizing behavior problems. MRI studies have shown that externalizing problems are characterized by a smaller or thinner PFC and ACC that may result from a developmental lag in growth. FMRI and TMS correlates of externalizing problems include less activity in the PFC and ACC during inhibitory and discrimination tasks. ERP correlates of externalizing problems that may tap into the hypoactivation in PFC and ACC regions include smaller amplitudes and longer latencies of the no-go N2 and the oddball P3. EEG correlates of externalizing problems include left frontal hemispheric asymmetry

(greater left than right frontal activity) in infant alpha frequency bands and less frontal infant alpha power. Time-frequency analyses find that externalizing problems, alcoholism in particular, may exhibit less frontal theta and parietal delta activity after an oddball target stimulus around the timing of the P3.

1.3.3 Synthesis of Literature

As a general summary of neural mechanisms in self-regulation and externalizing problems, there is evidence of the role of the PFC and ACC in behavioral regulation. Notably, neural differences in externalizing problems, both in terms of structure and function, have been observed in the PFC and ACC. Structurally, evidence typically suggests that smaller or less cortical thickness in the PFC or ACC is associated with poorer self-regulation and more externalizing problems. Functionally, evidence typically suggests that less activity or less efficient activity in the PFC or ACC during inhibition tasks (and the corresponding N2) is associated with poorer response inhibition and more externalizing problems. An altered attention network (and the corresponding P3 along with frontal theta activity) is also associated with poorer sustained attention and externalizing problems. The smaller amplitudes and longer latencies of the N2 and P3 may reflect less capable and efficient inhibitory control and sustained attention. These interpretations are bolstered by findings that (1) early environmental deprivation results in changes to PFC and ACC functioning (as indexed by the N2) in ways that are consistent with poorer response inhibition and (2) pharmacological and behavioral treatments for externalizing problems work by altering the N2 and dorsal ACC function, which has been linked to cognitive control.

In synthesis, structural and functional deficits in the PFC and ACC may map onto the behavioral dysregulation observed in externalizing problems. The neurophysiological correlates from different methods (MRI, fMRI, EEG) and analyses (ERP, EEG power analysis, time-frequency analysis) may reflect the same underlying neural process related to externalizing problems. In young children with externalizing problems, there may be a developmental lag in the growth of the PFC and ACC, which may result in hypoactivation of the PFC and ACC during tasks of inhibition and discrimination and may be manifested as (1) smaller amplitudes of the no-go N2 and oddball P3 and (2) less frontal theta activity after an oddball target stimulus around the timing of the P3. It is difficult to reconcile the fMRI finding of frontal hypoactivation with EEG findings of frontal hyperactivation (i.e., less alpha power) in the infant alpha frequency band. Several possibilities may explain the seemingly contradictory findings. First, EEG measured from topographically-frontal electrodes may not correspond solely to frontal brain regions. Second, EEG and fMRI measure different time scales, with fMRI limited to measuring relatively low-frequency activation. Speculatively, children with externalizing problems may show greater activation in alpha bands and less activation in lower frequency bands (e.g., delta and theta), which would be consistent with fMRI findings of frontal hypoactivation at lower frequencies. Third, the association between frontal activation and externalizing behavior may be nonlinear, as discussed earlier. Nevertheless, given the heterogeneous presentations of ADHD and externalizing problems, the cognitive and behavioral deficits likely reflect a distributed system of brain regions that differs between children.

Based on the neural correlates of externalizing problems identified in previous studies of older children and adults, the present study applies EEG/ERP methodology to extend

our understanding of the neural mechanisms in the development of externalizing behavior problems in very young children. Based on evidence that the N2 and P3 ERPs (that purportedly index functioning in the PFC and ACC among other regions related to inhibition and attention) are associated with externalizing problems, here we review the effect sizes from prior studies to determine the sample size necessary for adequate power to detect an association between ERPs and self-regulation and externalizing behavior problems.

A previous meta-analysis of studies examining the P3 in relation to externalizing problems in adults found a small effect size for smaller P3 amplitudes ($d = 0.25$) and longer P3 latencies ($d = 0.13$; Gao & Raine, 2009). We reviewed 16 studies that provided adequate information to calculate effect size estimates of the association between the N2 ERP and self-regulation or externalizing-related phenotypes in children. See Table 1.1 for the effect size estimates in each study. We calculated the average effect size by calculating the average of each study's effect size that was weighted by the study's sample size. The 16 prior studies had a medium effect size ($d = 0.46$, $R^2 = .05$) on average (the conversion of Cohen's d to R^2 was based on Equation 1.4). In other words, the N2 explained about 5% of the variability in self-regulation and externalizing problems. With this effect size, a study would need a sample size of 167 or larger to have power of .80 or greater to detect an association between the N2 and self-regulation or externalizing problems. Notably, none of the studies reviewed here met this sample size. Another way of increasing power is by including repeated measures (B. O. Muthén & Curran, 1997). None of the studies reviewed, however, included repeated measures of both ERPs and self-regulation or externalizing problems. Thus, many of the prior studies appear to be under-powered to detect an association between the N2 and self-regulation and externalizing problems. The finding that studies are under-powered

is common in neuroscientific investigations, and calls into question the validity of previous findings examining the N2 in relation to self-regulation and externalizing problems (Button et al., 2013; Jonas & Markon, 2014).

1.3.4 Limitations of Prior Studies

The previous research examining the neural correlates of externalizing behavior problems is characterized by key limitations. The vast majority of previous research is on older children and is cross-sectional. With one exception (C. L. Smith & Bell, 2010), neurophysiological research has not studied externalizing problems in children under age 5 or the longitudinal development of later behavior problems. Previously identified neural correlates of alcoholism and other externalizing problems in adults could reflect the effects of substance use and other antisocial behavior, and may not be causally related to the development of externalizing behavior. Because previous research on neural predictors has not examined (a) young children before behavior problems are fully established or (b) whether the neural risk factors lead to the development of later behavior problems, it is not known whether observed concurrent neural correlates are mechanisms in the development of externalizing problems. Understanding the mechanisms through which externalizing problems develop is crucial to isolate causal processes and to identify when and how externalizing problems arise. Externalizing problems in young children strongly predict later, more severe problems, including delinquency (Moffitt, 1990) and substance use disorders (Chilcoat & Breslau, 1999). The present study examines children from 30 to 42 months of age, a period when neural development is especially pronounced (Posner & Rothbart, 2000), externalizing problems are elevated (Tremblay, 2002), and when individual rank-order position on externalizing

TABLE 1.1: Effect Sizes of Previous Studies of N2 ERP Amplitudes and Latencies in Relation to Self-Regulation and Externalizing-Related Phenotypes.

Study	Task	Ages	ERP	Phenotype	Cohen's d	F	r	t	η^2	n_1	n_2
Albrecht et al. (2005)	stop-signal	8–14	N2 amp	ADHD	1.15					10	11
			N2 amp	ADHD+ODD/CD	0.66					11	11
			N2 amp	ODD/CD	0.89					8	11
Cragg et al (2009)	go/no-go	7	N2 amp	response inhibition	1.26		.53			26	
		9	N2 amp	response inhibition	−0.24		−.21			30	
Dimoska, Johnstone, Barry, and Clarke (2003)	stop-signal	7–12	N2 amp	ADHD	1.26	9.60				13	13
Fallgatter et al. (2004)	go/no-go	7–11	N2 lat	ADHD	1.11			3.19		16	19
Grabell (2014)	go/no-go	3–5	N2 amp	externalizing problems	0.98	4.98				14	18
Johnstone, Barry, and Clarke (2007)	stop-signal	8–14	N2 amp	ADHD	0.90	4.10				12	10
Johnstone, Barry, Markovska, et al. (2009)	go/no-go	8–14	N2 amp	ADHD	0.66	4.10				20	20

Study	Task	Ages	ERP	Phenotype	Cohen's d	F	r	t	η^2	n_1	n_2
Lahat et al. (2010)	go/no-go	4–5	N2 amp	response inhibition	0.70		.33			37	
Overtoom, Verbaten, et al. (1998)	go/no-go	6–14	N2 amp	ADHD	0.32	0.77				16	16
			N2 amp	ADHD+ODD	1.07	4.53				"	6
Pliszka, Liotti, et al. (2000)	stop-signal	10–12	N2 amp	ADHD	1.37	8.50				10	10
			N2 amp	response inhibition (controls)	1.06		.47			"	"
			N2 amp	response inhibition (ADHD)	1.81		.67			"	"
Ramautar et al. (2004)	stop-signal	18–23	N2 amp	response inhibition	−1.93	24.27				14	14
			N2 lat	response inhibition	1.79	20.83				"	"
Ramautar et al. (2006)	stop-signal	18–24	N2 amp	response inhibition	−3.33	77.61				15	15
			N2 lat	response inhibition	1.80	22.67				"	"
Schmajuk et al. (2006)	stop-signal	19–25	N2 amp	response inhibition	1.05	5.54				11	11
van Boxtel et al. (2001)	stop-signal	19–28	N2 amp	response inhibition	1.03	4.79				10	10
Wild-Wall et al. (2009)	flanker	11–17	N2 amp	ADHD	0.93	5.30				15	12
Woltering et al. (2011)	go/no-go	8–12	N2 amp	externalizing problems	−0.73	9.34			.10	71	24

Note. F, r, and t values were converted to Cohen's d for comparison and averaging (see Equations 1.1, 1.2, and 1.3). "n_1" refers to the sample size of group 1 (typically the clinical group or, if only one group, the entire sample). "n_2" refers to the sample size of the comparison group (if applicable, typically controls). "amp" refers to ERP peak amplitude. "lat" refers to ERP peak latency. Cohen's d estimates are recorded so that positive values reflect smaller amplitudes or longer latencies in self-regulation and externalizing problems (i.e., consistent with hypotheses). Negative values reflect larger amplitudes or shorter latencies in self-regulation and externalizing problems (i.e., counter to hypotheses). For studies that reported mean and standard

deviation of ages rather than a range of ages, a range was calculated as: mean ± 1 standard deviation. The equations used to convert parameter estimates to Cohen's d and r are below:

Converting F to d (Thalheimer & Cook, 2002):

$$d = \sqrt{F\left(\frac{n_1 + n_2}{n_1 \times n_2}\right)\left(\frac{n_1 + n_2}{n_1 + n_2 - 2}\right)} \tag{1.1}$$

Converting r to d (Dunst, Hamby, & Trivette, 2004):

$$d = \frac{2r}{(\sqrt{1 - r^2})} \tag{1.2}$$

Converting t to d (Dunst et al., 2004):

$$d = \frac{t(n_1 + n_2)}{(\sqrt{df})(\sqrt{n_1 \times n_2})} \tag{1.3}$$

Converting d to r (Borenstein, 2009):

$$r = \sqrt{\frac{d^2}{d^2 + 4}} \tag{1.4}$$

problems begins to stabilize (S. B. Campbell, Shaw, & Gilliom, 2000). EEG and ERPs are used in the present study to shed light on the neurodevelopmental process of externalizing disorders.

C. L. Smith and Bell (2010) argued that longitudinal studies of neural processes in early childhood are needed to identify externalizing psychopathology at earlier, more treatable stages. The proposed research aims to identify mechanisms in the development of externalizing problems from 2 1/2 to 3 1/2 years of age. Improved understanding of early predictors will ultimately improve early identification and intervention (Insel, 2014). Understanding the neural processes of externalizing problems in young children will yield a deeper understanding of the developmental mechanisms in psychopathology. The identification of developmentally meaningful early endophenotypes of externalizing problems may provide useful targets for treatment. A National Research Council (NRC) and Institute of Medicine (IOM) taskforce recommended that the National Institutes of Health (NIH) study neural risk factors for behavior problems in order to tailor preventive interventions to individuals at greatest risk (NRC & IOM, 2009). In addition, because younger children have greater neuroplasticity (Cramer et al., 2011), there is greater potential for prevention and treatment of externalizing psychopathology in young children (NRC & IOM, 2009; Stormont, 2002). Although externalizing disorders are often difficult to treat, prevention efforts targeted to younger, at-risk children have been more efficacious and cost-effective in altering later trajectories (e.g., Bierman, Coie, et al., 2007), with benefit-cost ratios of early childhood interventions about 8–9 to 1 (Heckman & Masterov, 2007). Thus, earlier identification of at-risk children may lead to more successful and cost-effective treatments, resulting in better self-regulation (Blair & Diamond, 2008; Diamond, Barnett, Thomas,

& Munro, 2007) and school readiness (Bierman, Domitrovich, et al., 2008), lower rates of substance use, delinquency, and medical problems, and a better quality of life for the individual, the family, and society in general (Moffitt et al., 2011). The proposed research takes an important step toward these public health goals by applying basic developmental and cognitive neuroscience techniques refined in older populations in order to identify developmental mechanisms in externalizing problems.

1.3.5 Why ERPs?

Mental and behavioral disorders are linked to abnormal brain development (Insel, 2014). Measures of neural functioning, ERPs in particular, may be especially informative for identifying developmental mechanisms and predicting later behavior problems before other behavioral risks can be detected. ERPs are likely more sensitive than behavioral measures for predicting later externalizing problems: (1) They are endophenotypes, closer to the genotype than behavior, summing genetic and environmental influences (Loo, Lenartowicz, & Makeig, in press). Neural measures will likely improve our detection of genetic liabilities for psychological disorders (Glahn, Thompson, & Blangero, 2007). (2) The same behavior can arise from different underlying brain activity patterns (e.g., Dimoska & Johnstone, 2007) and can reflect different trajectories of psychopathology. Behavioral symptoms are imprecise and provide little information about the underlying mechanisms (Insel, 2014). Neural measures can distinguish underlying reasons for behavior, and may provide more accurate and specific diagnosis of disorder subtypes (Haubold, Peterson, & Bansal, 2012). Researchers have identified potential neurophysiological subtypes of ADHD using EEG (Barry, Clarke,

McCarthy, & Selikowitz, 2003; Clarke & Barry, 2004; Clarke, Barry, McCarthy, & Selikowitz, 2001; Clarke, Barry, McCarthy, Selikowitz, & Brown, 2002; Kirk, 2007; Mazaheri, Fassbender, Coffey-Corina, Hartanto, Schweitzer, & Mangun, 2014). MRI (Bansal et al., 2012; Hart et al., 2014; Lim et al., 2013) and EEG (Magee, Clarke, Barry, McCarthy, & Selikowitz, 2005) measures have shown sensitivity and specificity in diagnosing ADHD (but the clinical utility of using neural measures to diagnose ADHD has been questioned based on the low base rate of ADHD; Willis & Weiler, 2005). ERP measures have shown fairly good accuracy in diagnosing ADHD subtypes (J. L. Smith, Johnstone, & Barry, 2003). Moreover, a study showed that EEG measures have as much sensitivity as, and greater specificity than, standard psychiatric evaluations for diagnosing ADHD (Quintana, Snyder, Purnell, Aponte, & Sita, 2007). (3) Behavioral symptoms are late manifestations of the underlying brain process (Insel, 2014). Neurophysiology likely demonstrates risk prior to behavior demonstrating the risk in clearly disordered behavior. Neural measures have shown abnormalities that precede the onset of clinical symptoms (Haubold et al., 2012). (4) Behavioral performance measures may reflect compensation processes, such as momentary high motivation, but ERP indexes of brain processing inefficiency may nevertheless identify risk for dysregulation and maladjustment. Supporting the incremental predictive utility of brain functioning, less go/no-go ACC activity predicted later recidivism in criminals over and beyond other risks (Aharoni et al., 2013). Moreover, neuropsychological profiles that included brain structure and function (such as failed inhibition in a stop-signal task) as measured by fMRI in addition to cognitive, personality, genetics, history, and demographics domains accurately classified (AUC = .96) and predicted later (AUC = .75) binge drinking among adolescents (Whelan et al., 2014). These neural processing measures may be even

more important in early childhood when behavioral indicators are less developed. Two examples of ERPs as better predictors of later behavioral disorders than early behavior measures are dyslexia and autism: infants' ERPs predicted later dyslexia with sensitivity and specificity at age 8 (Molfese, 2000) and later autism diagnosis at age 3 (Elsabbagh et al., 2012). In both cases, the ERPs successfully predicted later disorders at ages before behavioral measures are useful. ERPs are an ideal tool for identifying neural mechanisms in externalizing problems because ERPs are sensitive to variations in cognitive and motor processes in addition to pharmacological manipulations (Soltani & Knight, 2000) and genetic risk for externalizing problems (Hicks et al., 2007; Iacono & Malone, 2011). As a result, ERPs may be useful for probing disorders of altered neurotransmission.

Early neural markers may be especially sensitive to risk for developing later externalizing problems because the effects of neural processing inefficiency on behavior may compound with age as children experience developmental challenges, and have greater capacity and more opportunities to exhibit externalizing behavior. Externalizing problems are thought to reflect abnormal stimulus processing (Crozier et al., 2008; Fite, Goodnight, Bates, Dodge, & Pettit, 2008; Lansford, Malone, Dodge, Crozier, Pettit, & Bates, 2006) and "acting without thinking" (Romer, 2010, p. 265), so ERPs may be particularly informative because of their high temporal resolution, allowing examination of brain activity during early stimulus processing (e.g., Molfese, Key, et al., 2006). Consequently, ERPs should be useful in detecting differences in processing environmental input and in predicting poor behavioral response. For these reasons, ERPs may be more sensitive to later externalizing problems than traditional measures, and may allow earlier, more accurate detection and earlier intervention or

even prevention. Ultimately, we do not view ERPs as a replacement to traditional behavioral measures of risk, but rather as an important supplement. Successful early identification of at-risk children will require better early behavioral and neural measures of risk. Neural measures are useful for understanding the developmental processes by which genetic and environmental factors combine and interact to develop behavior problems. In sum, ERPs provide an excellent chance to predict later externalizing problems successfully from a very early age, and to identify early developmental mechanisms in externalizing problems that may be more easily treated.

1.3.6 Behavioral Mechanisms

The proposed study also considers possible behavioral intermediate phenotypes that mediate the neural risk factors on the development of externalizing problems in order to advance understanding of the developmental mechanisms in externalizing psychopathology. For example, the present study includes neural and behavioral measures of self-regulation, which has been considered an intermediate phenotype of externalizing behavior (Calkins & Howse, 2004; Doyle et al., 2005; Gagne et al., 2011; Hardaway et al., 2012; Olson, Sameroff, et al., 2005; Patrick, Venables, et al., 2013; Slaats-Willemse et al., 2003; Sulik et al., in press; Vijayakumar et al., 2014; Young et al., 2009), and may reflect a key mechanism by which etiological factors influence externalizing behavior. Indeed, a prior study showed that changes in self-regulation mediated the effects of ACC cortical thinning on the development of externalizing problems (Vijayakumar et al., 2014). Moreover, self-regulation deficits are characterized by deficits in the cognitive components presumably measured by the N2 and

P3 (e.g., inhibitory control, sustained attention), so it is likely that many domains of self-regulation covary with the N2 and P3, as supported by prior findings (Wiersema & Roeyers, 2009), and mediate the effects of the N2 and P3 on externalizing. Thus, self-regulation deficits may reflect shared neural deficits among many forms of externalizing psychopathology and may account for their comorbidity. As a result, self-regulation deficits may be informative for transdiagnostic classification of externalizing disorders (Nolen-Hoeksema & Watkins, 2011; Patrick, Venables, et al., 2013).

1.4 The Present Studies

This report details two studies of the relation between neurophysiology, self-regulation, and externalizing behavior problems in very young children.

1.4.1 Purpose

The purpose of the studies is to determine the neural mechanisms underlying the development of externalizing behavior problems in order to (a) promote earlier identification of children at risk for developing later behavior problems and (b) understand the processes by which externalizing problems develop, which could illuminate useful targets for intervention.

1.4.2 Outline of Studies

In Study 1, we used a go/no-go task to measure neural components reflecting inhibitory control and a P3b oddball task requiring sustained attention. To include most children in the analyses, we included incorrect trials in the ERP averages (for a more detailed

rationale, see Section 2.1.4). In Study 2, we used task paradigms and procedures that are more developmentally appropriate for young children, including a more child-friendly go/no-go task with performance feedback and a P3a oddball task that does not require a behavioral response on the part of the child. Through pilot work, the more developmentally-appropriate nature of the go/no-go task in Study 2 elicited an adequate number of correct trials from most children in both go and no-go task conditions. Thus, unlike Study 1, we included only correct trials in the EEG/ERP analyses in Study 2. We followed the children longitudinally to determine whether the neurophysiological components of interest predict later changes in self-regulation and externalizing problems.

1.4.3 Hypotheses

Based on findings from previous studies, the present studies tested 7 hypotheses that **more externalizing problems, both concurrently and longitudinally, would be associated with:**

Hypothesis 1. Smaller amplitudes of the no-go N2.

Hypothesis 2. Longer latencies of the no-go N2.

Hypothesis 3. Smaller amplitudes of the oddball P3 (P3b in Study 1, P3a in Study 2).

Hypothesis 4. Longer latencies of the oddball P3 (P3b in Study 1, P3a in Study 2).

Hypothesis 5. Left frontal asymmetry (greater activation at left than right frontal electrodes) in infant alpha (6–9 Hz) frequency bands.

Hypothesis 6. Less frontal infant alpha power.

Hypothesis 7. Less frontal theta activity, particularly during the timing of the no-go N2 and oddball P3.

It was also hypothesized that neurophysiology would predict self-regulation:

Hypothesis 8. The N2 would predict deficits of disinhibition and aggression.

Hypothesis 9. The P3 (particularly the P3b) would predict sustained attention deficits.

Moreover, for those neurophysiological components associated with externalizing problems, it was hypothesized that:

Hypothesis 10. Self-regulation changes would mediate the effect of ERPs on the development of externalizing problems.

1.4.4 Innovation

Few studies have examined the neural correlates of externalizing problems in children under age 5, or whether neural risk factors predict the development of later behavior problems. The biggest innovation of the present study is that it examines whether neural biomarkers predict the development of later externalizing problems in young children. Identifying early biomarkers of externalizing is feasible; early ERP biomarkers have been detected for other disorders, including autism and dyslexia. Most neural studies investigating externalizing problems have focused on specific diagnoses, including ADHD, CD, and ODD. The present study of externalizing problems measured dimensionally should improve predictive precision (Persons, 1986), better account for the transdiagnostic genetically-based comorbidity of externalizing disorders (Patrick, Venables, et al., 2013), and lead to more refined treatments, because externalizing disorders are dimensional (Coghill & Sonuga-Barke, 2012; Krueger, Markon, Patrick, & Iacono, 2005; Markon & Krueger, 2005; Walton, Ormel, & Krueger, 2011). This study accords with recent calls to examine neurophysiological indicators of

dimensional models of externalizing problems (Patrick, Venables, et al., 2013). A relatively innovative sub-question is whether the N2 more strongly predicts inhibition deficits than does the P3, whereas the P3 more strongly predicts attention deficits than does the N2.

This study also incorporates more precise measurement of behavioral phenotypes for self-regulation, with multiple indicators. This accords well with the National Institute of Mental Health (NIMH) Research Domain Criteria (RDoC; NIMH, 2011) that seek to classify psychopathology across underlying, cross-cutting dimensions in order to identify endophenotypes and other risk factors that are more amenable for treatment. This study examines two important RDoC principles to better understand mechanisms in the development of externalizing psychopathology: (1) neurodevelopmental trajectories (examining the relation of brain and behavior across development) and (2) sensitive periods (examining when the brain and behavior are greatest influenced by experience; Casey, Oliveri, & Insel, 2014). The current study examines developmental change in self-regulation and externalizing behavior in relation to underlying brain changes in sustained attention and inhibitory control, and does this during a sensitive period of self-regulation development. Studies suggest that examining underlying phenotypes of psychopathology is useful. A meta-analysis found support for the endophenotype hypothesis that neurobiological phenotypes are better than trait or diagnostic measures for identifying the substrates of psychopathology because they are more proximal to the underlying etiology (Jonas & Markon, 2014). The study found that the strength of association between genotype and phenotype for the following phenotypes, ordered from largest to smallest association, was: (1) neurobiological, (2) neuropsychological, (3) trait, and (4) diagnostic phenotypes. Thus, examining the N2/P3 (neurobiological phenotype) and inhibitory control/sustained attention (neuropsychological) may be more

robustly associated with the underlying etiology of externalizing problems than are rating scales of externalizing problems (trait) or diagnoses of externalizing problems (diagnostic).

Chapter 2

Study 1

2.1 Method

2.1.1 Participants

Children and their families were recruited in the period April 2012 to September 2013 from the Bloomington, Indiana area to participate in a study with assessments of neural functioning, self-regulation, and behavior problems. Participants were found through a developmental research database and through a public housing agency. Of the 53 families, 45 (85%) agreed to participate in the EEG procedures. Of those who agreed to participate in the EEG procedures, 39 (87%) actually had an EEG visit (some families were unable to schedule a visit due to scheduling difficulties). Children were assessed with EEG procedures at 30 ($n = 33$), 36 ($n = 14$), and 42 ($n = 1$) months of age (this includes some children who were assessed at multiple occasions). Most children were assessed at 30 months of age because children were assessed as part of an ongoing longitudinal study, but we changed the ERP tasks (see Study 2) before following up some children until 42 months of age. Children were included in the analyses for the present report if they had usable EEG data

at one or more measurement occasions, resulting in a final sample of 27 unique children. In order to examine the co-development of brain functioning and behavior over time, some children ($n = 8$) had multiple EEG assessments, resulting in a final sample of $N = 35$ cases. Mean age at the EEG assessment was 33.53 months ($SD = 3.19$). Of the final sample, 12 (44%) children were girls, and 15 (56%) were boys. Although it would have been ideal to have longitudinal data on all children, using the available longitudinal data provides better power than using only one time point (and discarding the remaining data), given that we can account for dependency in the data.

Parents (usually the mother) reported on the child's behavior problems. Among the parent reporters, 25 (93%) were female, 92% were Caucasian, 4% were Asian-American, and 4% were of mixed race. Information on child ethnicity was not collected. There were 25 mothers, 2 fathers, and 96% were biological parents. Parents ranged in age from 25 to 49 years old ($M = 33.87$, $SD = 5.26$). Of parents, 96% were married, 4% were single, 94% had a college degree, and 4% had completed some college. The Hollingshead four-factor index of socioeconomic status (SES; Hollingshead, 1975) ranged from 13 to 66 ($M = 48.36$, $SD = 13.48$), suggesting a sample with some variation in SES, but with a solid middle-class core.

In addition to collecting parent reports of behavior problems, with the parents' permission, we asked secondary caregivers to rate behavior problems. Secondary caregivers were persons (over age 18) not living with the child who spent the most time with the child (and at least 10 hours) in the past 30 days. Parents did not name a secondary caregiver at ages 30, 36, and 42 months for 67%, 50%, and 0% of the children, respectively (only 1 child was assessed at 42 months of age). Of the children whose parents named a secondary caregiver,

40%, 67%, and 0% of their secondary caregivers participated at 30, 36, and 42 months, for a total of 21 secondary caregivers. Of these secondary caregivers, 74% were teachers, 11% were babysitters, and 11% had other connections to the child (however this was only based on 63% of the sample that was assessed under the protocol that included secondary caregivers).

2.1.2 Measures

For an adequate sample to determine the reliability and validity of measures, we calculated descriptive statistics and estimates of interrater reliability, cross-time continuity, internal consistency, and convergent and discriminant validity using the full sample of 336 unique families who were part of the larger study (i.e., not just those who were recruited for the EEG procedures). We also examined whether self-regulation and externalizing problems showed increasing stability over time in individual differerences. We tested whether cross-time continuity increased from 30–36-months to 36–42 months using Williams's test of two dependent correlations sharing one variable (i.e., measures at 30 and 42 months both shared associations with measures at 36 months; Steiger, 1980).

2.1.2.1 Neurophysiology

2.1.2.1.1 Electrophysiological Data Acquisition. Electrophysiological data have shown strong reliability in children (ERP: Hämmerer, Li, Völkle, Müller, and Lindenberger, 2013; Räikkönen, Birkás, Horváth, Gervai, and Winkler, 2003; EEG: Gao, Tuvblad, Raine, Lozano, and Baker, 2009; Vuga, Fox, Cohn, Kovacs, and George, 2008), and were collected via the Electrical Geodesics Inc. (EGI) 128-electrode EEG system with a Net Amps 300

series amplifier, which is accurate over a range of EEG frequencies (Ferree, Luu, Russell, & Tucker, 2001). The NetStation software controlled impedance measures, baseline correction, analog-to-digital sampling, data storage, artifact rejection, and signal averaging. The stimuli were presented in E-Prime 2.0 (Schneider, Eschman, & Zuccoloto, 2007), which interfaced with the NetStation 4.4.2 (Electrical Geodesics Inc., 2006) recording system. Visual stimuli were presented on a monitor that was 30 cm tall and 38 cm wide, at a distance of 1 meter from the child. Auditory stimuli were presented at 75 decibels using an 8 ohm speaker powered by an 80 watt amplifier. For a subset of participants, a speaker was placed on each side of the monitor, 1 meter from the child. After a change in protocol, the speaker was placed 1 meter above the center of the child's head. EEG signals were recorded at 4 ms intervals (250 Hz) with bandpass filters of 0.1 to 100 Hz while the experimenter continuously monitored the ongoing EEG signal. Testing was suspended during periods of movement artifacts and resumed after the artifact-free EEG returned to baseline for 2 seconds.

2.1.2.1.2 Electrophysiological Tasks. EEG activity and ERPs were recorded as toddlers participated in two age-appropriate self-regulation tasks, counterbalanced in order.

1. Sustained attention task, 6 minutes: an auditory oddball (two-sound discrimination) task requiring sustained attention was used to elicit a P3b (P300) ERP component to infrequent sounds (cat "meow" or duck "quack"). Sounds denoting the infrequent stimulus were counterbalanced across children. Prior to testing, the child was presented a series of practice trials. Children were trained to press a large green button when they hear the infrequent sound. Eighty sounds lasting 1 second occurred at 2.7-second intervals, giving

the child adequate time to respond. Sounds were randomly ordered so that one occurred 70% of the time and the other occurred on 30% of the trials, for a total of 56 frequent and 24 infrequent (target) trials. The child was instructed to press the large green button, with their preferred hand, only when the infrequent sound occurred. Participants had, on average, 9.57 ($SD = 2.76$) bad electrode channels during the task. Participants contributed 9.94 ($SD = 4.87$) usable target and 23.73 ($SD = 10.25$) usable frequent trials on average. Means and 95% confidence intervals (CIs) of behavioral performance (percent correct) on each trial condition are presented in Figure 2.1. Children responded to a greater percentage of the relevant target stimulus trials than the nontarget stimulus trials ($t[32] = 4.63$, $p < .001$), suggesting that children successfully categorized the stimuli.

2. Inhibitory control task, 6 minutes: a go/no-go task, in which the child was asked to follow directions from a bird picture (see Figure 2.2) and ignore those from the alligator picture (see Figure 2.3), was used to elicit a response inhibition potential (N2 or N200) on the no-go trials. Children were instructed to push the large green button when the bird says "Push," but not when the alligator says "Push." Stimuli were presented randomly so that the bird appeared on half of trials and the alligator on the other half. During picture presentation, children's ERPs were recorded to the animal's voice saying "push" 7 times at 1.7-second intervals. There were a total of 12 trials and 84 "push" commands (7 per trial), with 42 go and no-go trials each. Order of trials and go versus no-go animals were randomized across children. Each trial block lasted about 12 seconds, well within the attention span of toddlers. Intertrial intervals varied from 1.8 to 2.8 seconds to prevent habituation. ERPs were recorded to each "push" during a trial. These stimulus presentation procedures work well with very young children (Molfese, Morse, & Peters, 1990).

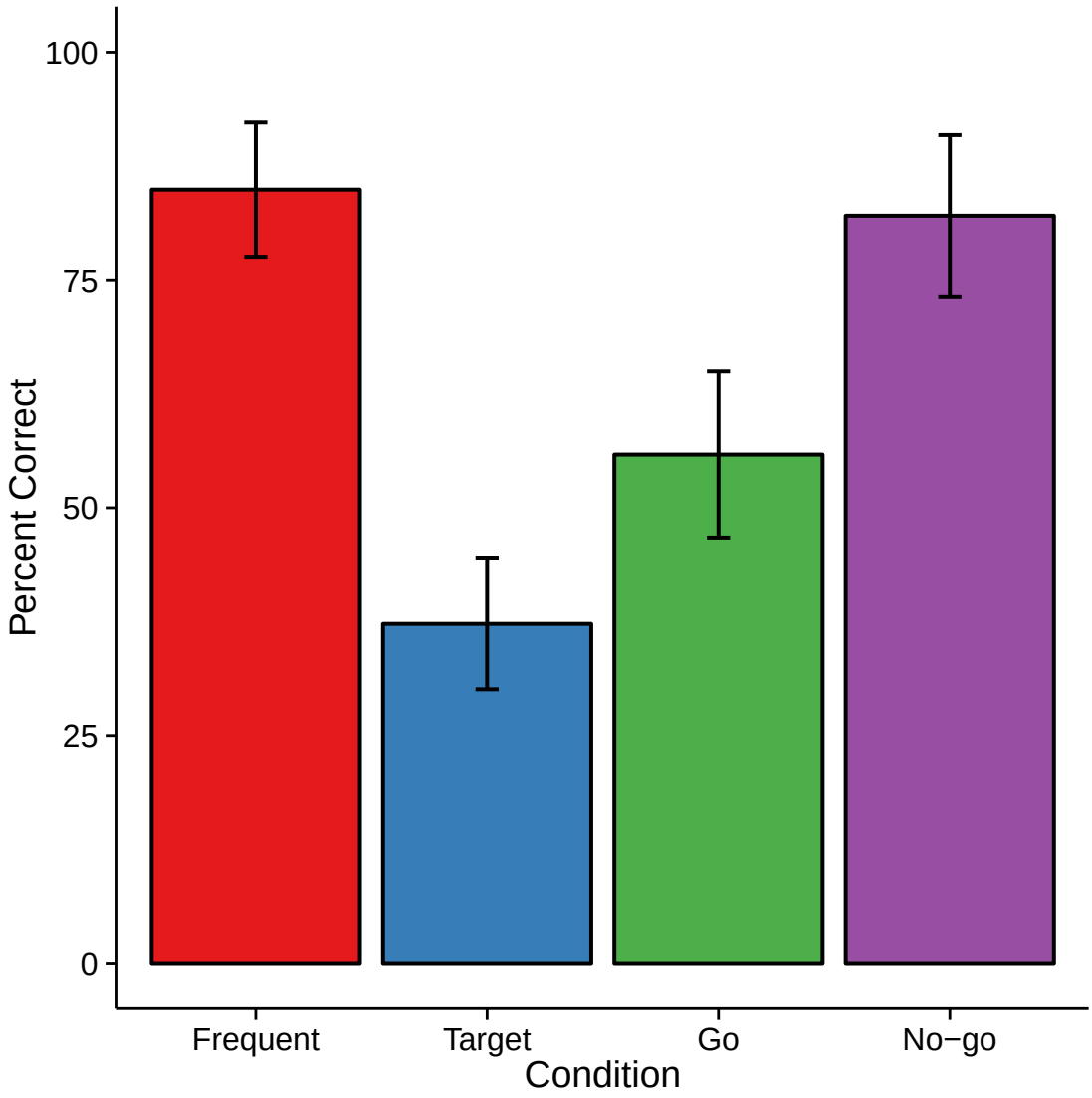

FIGURE 2.1: Bar graph of behavioral performance (percent correct) in auditory oddball and Bird/Alligator electrophysiological tasks. Error bars represent 95% confidence intervals.

Participants had, on average, 10.08 ($SD = 3.36$) bad electrode channels during the task. Participants contributed 18.76 ($SD = 8.18$) usable go and 19.24 ($SD = 7.43$) usable no-go trials on average. Means and 95% CIs of behavioral performance (percent correct) on each trial condition are presented in Figure 2.1. Children responded more frequently to the relevant go stimulus than the no-go stimulus ($t[36] = 7.53$, $p < .001$), suggesting that children successfully categorized the stimuli and were fairly successful at selectively responding to the go stimulus.

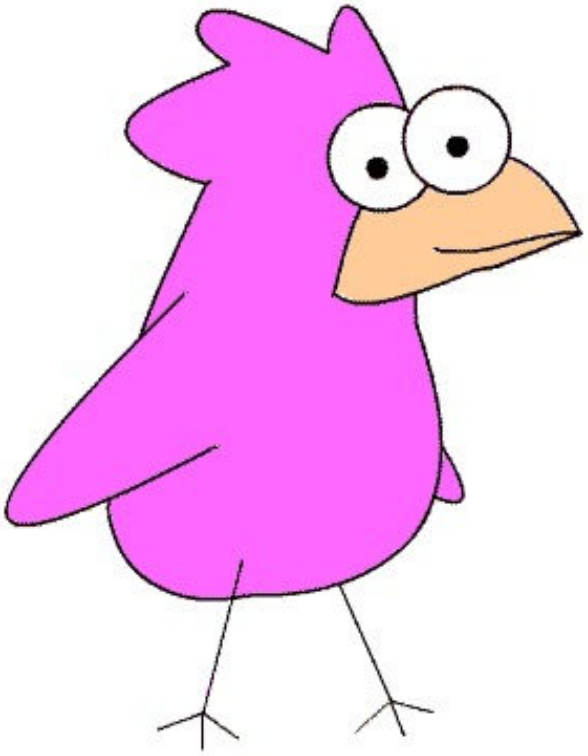

FIGURE 2.2: Picture of Bird from Bird/Alligator ERP task.

There were offsets between E-Prime's command to present the stimuli and the actual presentation of the auditory stimuli through the speakers in both tasks. Offsets were approximately 4 ms, on average, for the auditory cat meow and duck quack sounds in the

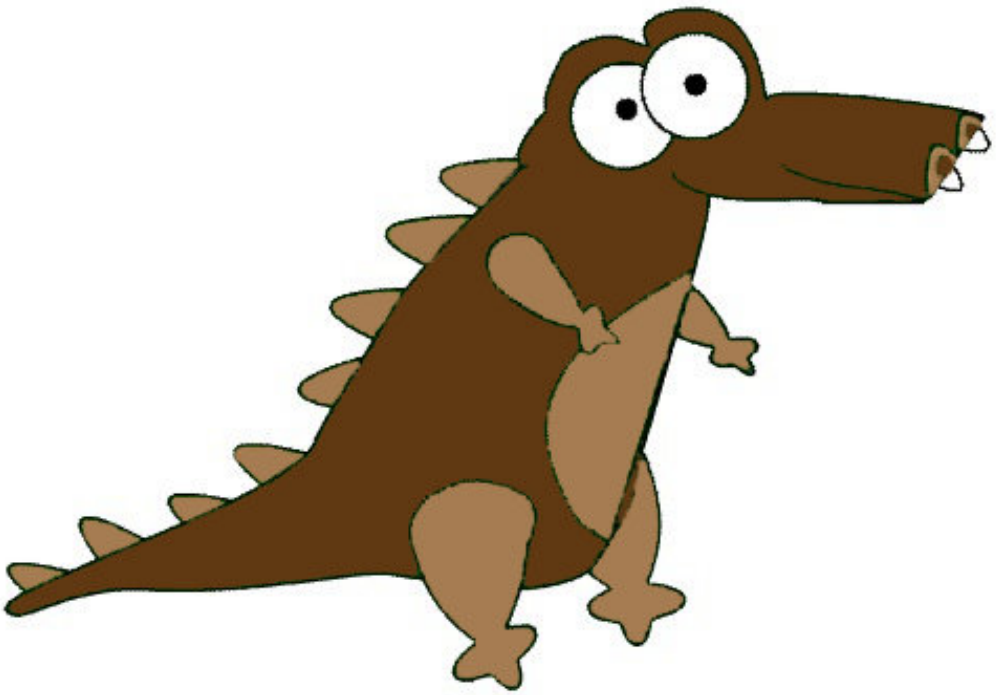

FIGURE 2.3: Picture of Alligator from Bird/Alligator ERP task.

oddball task. Offsets were approximately 5 ms, on average, for the alligator "push" command and 10 ms, on average, for the bird "push" command in the Bird/Alligator task. There were also analog-to-digital conversion delays of 8 ms for the EGI Net Amps 300 series amplifier. These offsets and delays were accounted for by shifting the windowed time frame later during trial segmentation by the sum of the offset and the 8 ms analog-to-digital conversion delay. In addition to the two tasks, for some children ($n = 15$), 5 minutes of baseline EEG were recorded while children watched a child-friendly video they chose. The baseline data were not examined in this study.

Prior studies have shown that meaningful findings can be elicited from fewer than 10 ERP trials (e.g., Stets & Reid, 2011). As a result, we examined the tradeoff between (a) the number of participants retained at different cutoffs for number of good (artifact-free) trials and (b) the quality of the resulting ERP waveforms. For a given task, we decided to retain participants who had at least 9 good trials in both conditions, in order to retain the most possible participants while ensuring a good quality of data. Thus, to have usable data, we set a cutoff of at least 100 good channels and 9 good trials in both conditions. More than half of EEG assessments (60%) yielded usable data, in line with prior studies of this age (Bell & Cuevas, 2012). Reasons for EEG missingness included: did not wear cap (8%), refused to play (16%), too many bad trials (i.e., fewer than 9 good trials in any condition; 14%), and other technical problem (2%).

2.1.2.1.3 ERP Data Processing. To examine the hypotheses of smaller amplitudes and longer latencies of the N2 and P3 in self-regulation and externalizing problems, we processed the data for ERP analysis in the following order:

1. Signals were filtered using a 30 Hz digital low pass filter and a 0.3 Hz high pass filter.

2. The ERP data were epoched into separate trials as a function of experimental condition (target versus nontarget, go versus no-go). Segmentation involved a 200 ms prestimulus and 1000 ms poststimulus period.

3. Clearly bad channels were manually rejected.

4. Manually specified bad channels were interpolated based on the waveforms of surrounding channels.

5. Artifacts and bad channels were rejected on a trial-by-trial basis using the following criteria:

    - Bad channels: maximum amplitude minus minimum amplitude exceeded 150 μV during any 80 ms moving average of the segment. A channel was marked bad for the entire task if it was marked bad for greater than 30% percent of trials.
    - Eye blinks: maximum amplitude minus minimum amplitude exceeded 150 μV during an 80 ms moving average of a 1000 ms window during the segment.
    - Eye movement: maximum amplitude minus minimum amplitude exceeded 150 μV during an 80 ms moving average of a 1000 ms window during the segment. Although it is an assumption that a 150 μV change would reflect an eye blink or movement, we applied a threshold that is common in standard electrophysiological processing.
    - Bad trial: Contains more than 20 bad channels, or an eye blink or eye movement.

6. Bad channels were interpolated based on the waveforms of surrounding channels.

7. Data were re-referenced to an average reference so that each electrode's signal reflected its deviation from the average signal of all other electrodes.

8. The data epochs were baseline corrected (i.e., subtracting out the mean of the baseline period), using the average of the 200 ms prestimulus period. After the above steps were applied to individual cases, we applied data reduction techniques on the collective sample:

9. Data were reduced using principal components analysis (PCA; see below).

Data reduction involved a sequential PCA to account parsimoniously for the majority of variability in the waveform using the ERP PCA Toolkit version 2.47 (Dien, 2010) for MATLAB version 7.14.0.739 (The MathWorks Inc., 2012): (1) A temporal PCA with promax rotation identified time factors. (2) A separate spatial PCA with infomax rotation was then conducted on every time factor to allow each time factor to be characterized by different spatial topographies. The number of factors to retain was selected objectively by comparing a scree plot to random noise, and retaining the number of components whose eigenvalues exceeded that of random data (Dien & Frishkoff, 2005). The temporospatial PCA seeks to distinguish the different underlying ERP components (that are thought to reflect different cognitive processes; see Figure 2.4). The PCA components corresponding to the timing and spatial topography of the N2 and P3 (reflecting variability in N2 and P3 amplitudes) were used as predictor variables in the hypothesized models to determine if individual differences in ERP amplitudes are associated with individual differences in externalizing behavior. To identify individuals' ERP latencies, the latency of the peak amplitudes for the N2 and P3 were identified for each individual given their scores on the spatial PCA components.

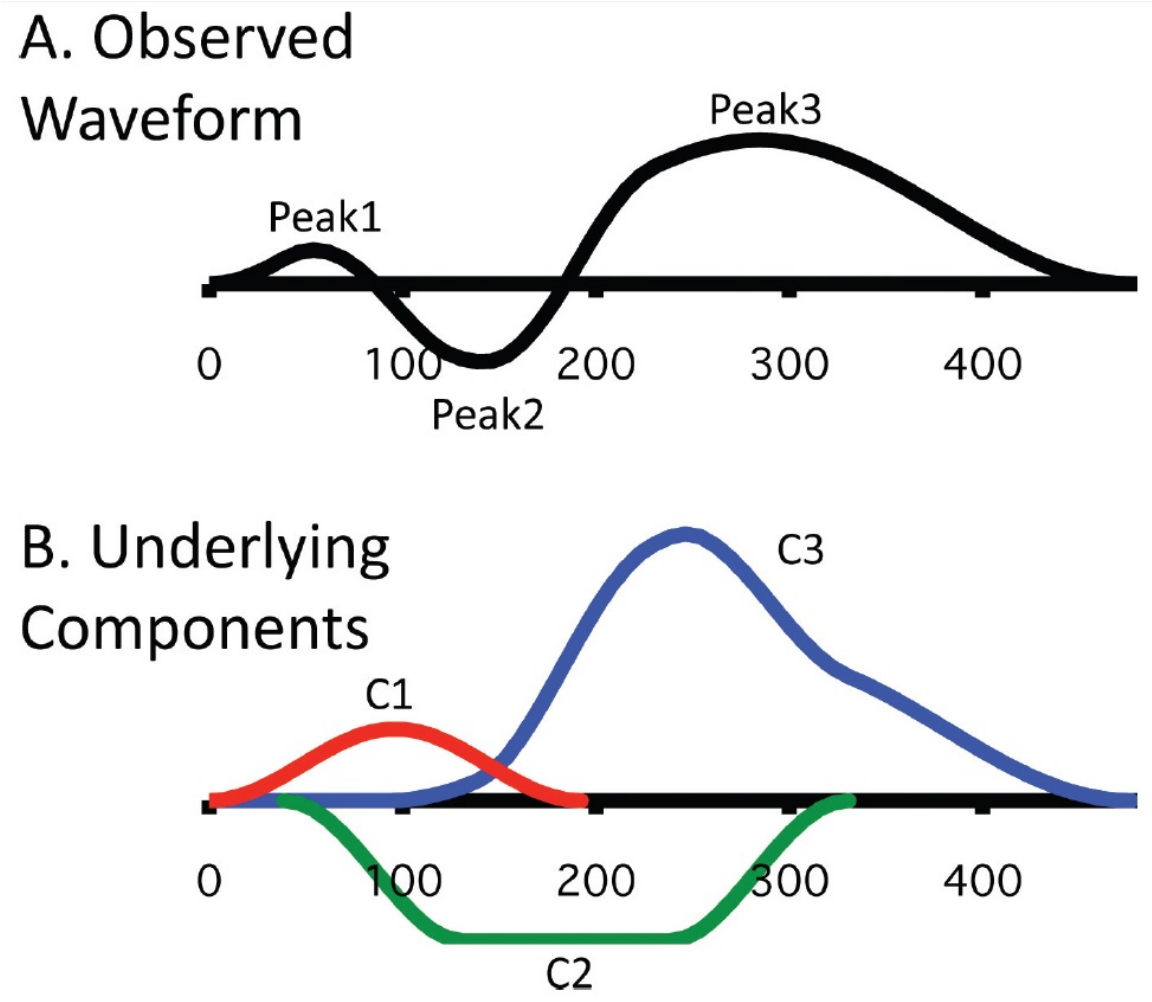

FIGURE 2.4: The underlying ERP components (Panel B) sum together to produce the observed ERP waveform (Panel A). The temporospatial PCA seeks to distinguish the different underlying ERP components (that are thought to reflect different cognitive processes). Printed from Kappenman and Luck (2011) with permission from Oxford University Press.

A topo plot depicting the ERP waveforms grand-averaged across participants at each electrode site for the go and no-go conditions of the Bird/Alligator ERP task is in Figure 2.5. Grand-averaged ERP waveforms averaged across frontocentral electrodes for the go and no-go conditions are in Figure 2.6. A spatial PCA identified 13 spatial components (i.e., independent electrode clusters) accounting for 87% of the variance in the ERP waveform. A separate temporal PCA identified 12 temporal components (i.e., independent time windows)

accounting for 95% of the variance. A spatial PCA on the ERP data reduced by the temporal PCA (i.e., a temporo-spatial PCA) identified 6 spatial components accounting for 80% of the variance. The peak latency of the temporo-spatial component corresponding to the N2 was 572 ms (see Figure 2.7). The electrode cluster for the frontocentral spatial component corresponding to the N2 is displayed in Figure 2.8. The peak latency of the frontocentral spatial component corresponding to the N2 was 574 ms (see Figure 2.9). To identify each individual's peak latency of the N2 component, we identified each individual's latency to their peak minimum amplitude (because the N2 is a negative-going component) during the range of 574 ± 50 ms: 524–624 ms.

Descriptive statistics of children's N2 amplitudes and latencies are in Table 2.1. Pearson correlations of children's N2 amplitudes and latencies are in Table 2.2. The N2 and P3 were not strongly correlated, which is unsurprising because they were measured using different paradigms and therefore have different meanings from the N2-P3 complex measured in the same paradigm (although we did not observe both N2 and P3 components in the same paradigm). Nevertheless, children with larger (more positive) P3 amplitudes tended to have smaller (less negative) N2 amplitudes. Children's no-go N2 amplitudes tended to be larger (i.e., more negative) than their go N2 amplitudes ($t[33] = -2.00$, $p = .054$), providing some evidence that the no-go N2 may be related to inhibitory processing. Among the 7 children with usable ERP data on the Bird/Alligator task at multiple assessments (out of 8 children with multiple EEG assessments), the cross-time continuity of the N2 amplitude was $r(5) = .12$ ($p = .790$) and N2 latency was $r(4) = .80$ ($p = .058$). This suggests stronger cross-time continuity of N2 latencies than amplitudes. Correlations between the N2 and child's age were $r(32) = -.14$ ($p = .444$) for N2 amplitudes and $r(31) = -.11$ ($p = .536$) for

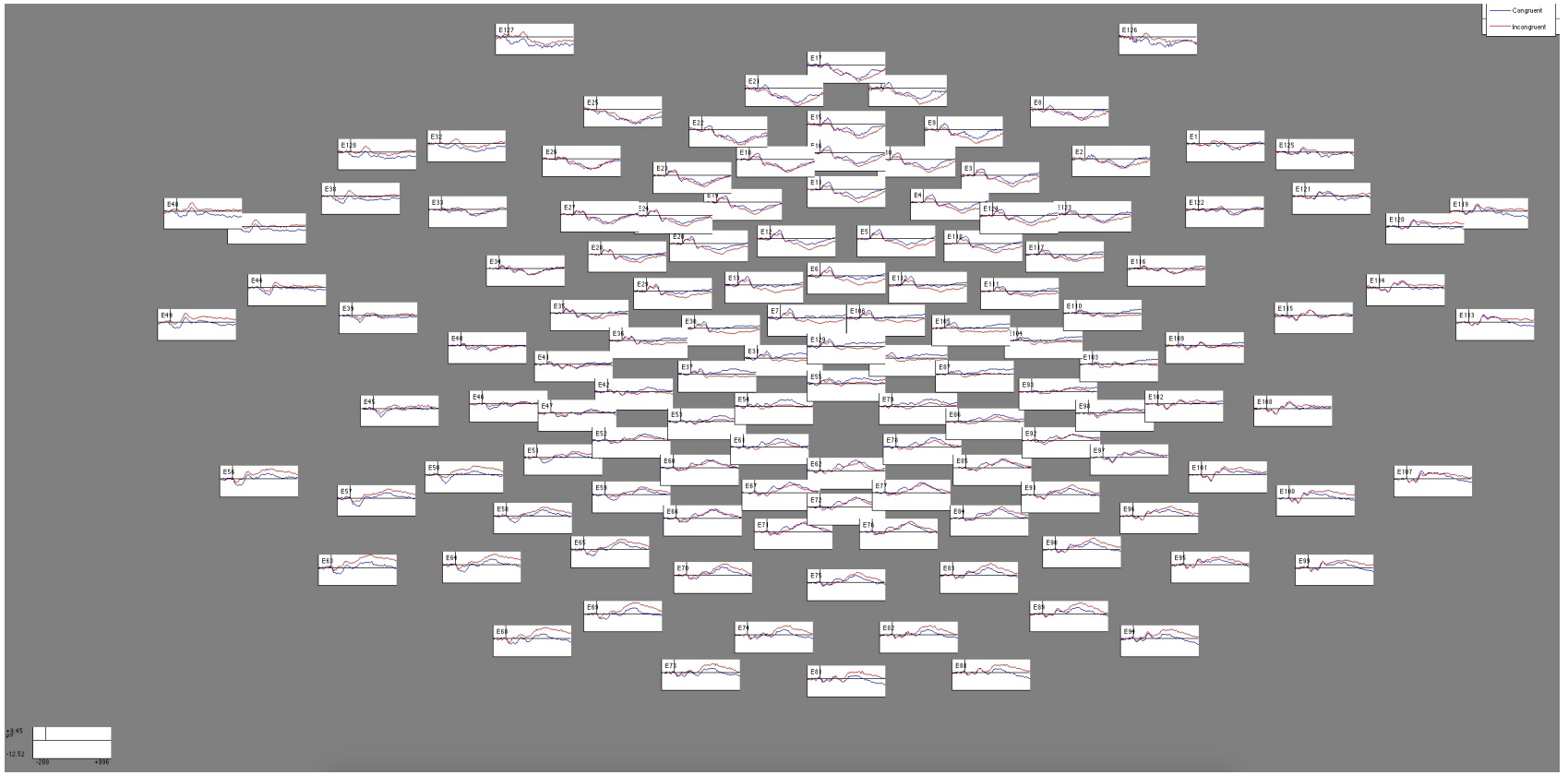

FIGURE 2.5: Topo plot of grand-averaged ERP waveforms for the go (blue) and no-go (red) conditions of the Bird/Alligator ERP task. The negative-going wave corresponding to the N2 can be observed in the frontocentral electrodes. Frontal electrodes (e.g., E17) are located at top of diagram, posterior electrodes (e.g., E81) at bottom.

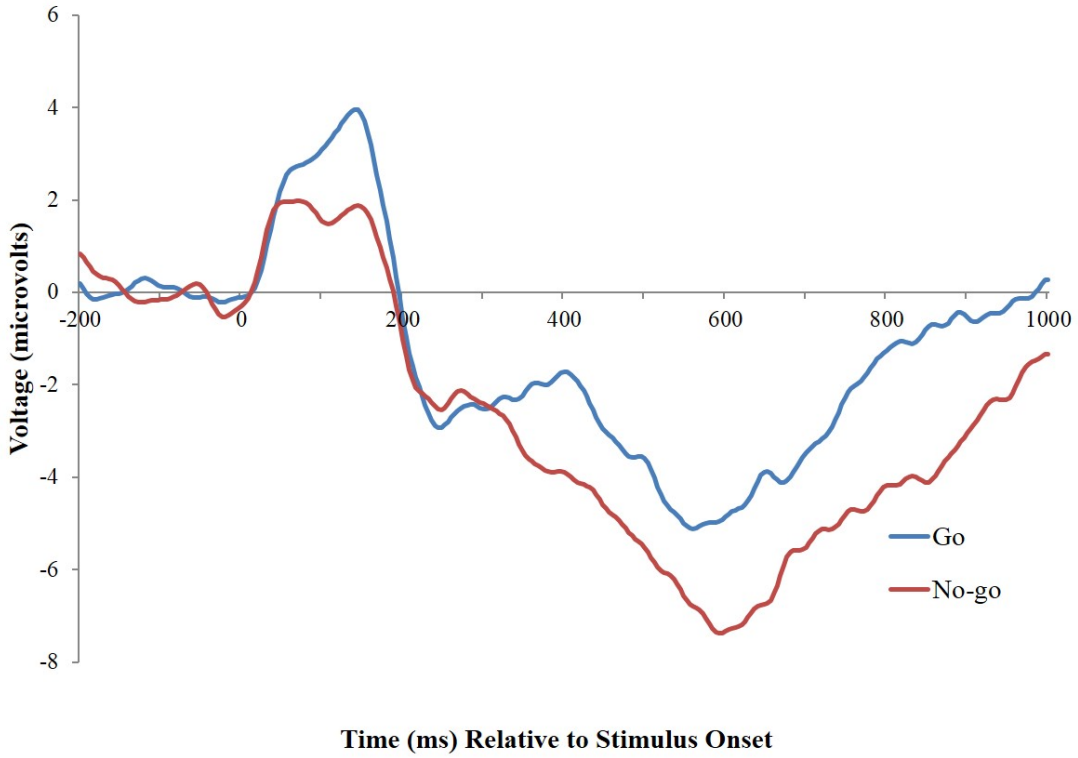

FIGURE 2.6: Grand-averaged ERP waveforms averaged across frontocentral electrodes for the go and no-go conditions of the Bird/Alligator ERP task. Waveforms were averaged across electrodes from the frontocentral electrode cluster identified by the spatial PCA (see Figure 2.8).

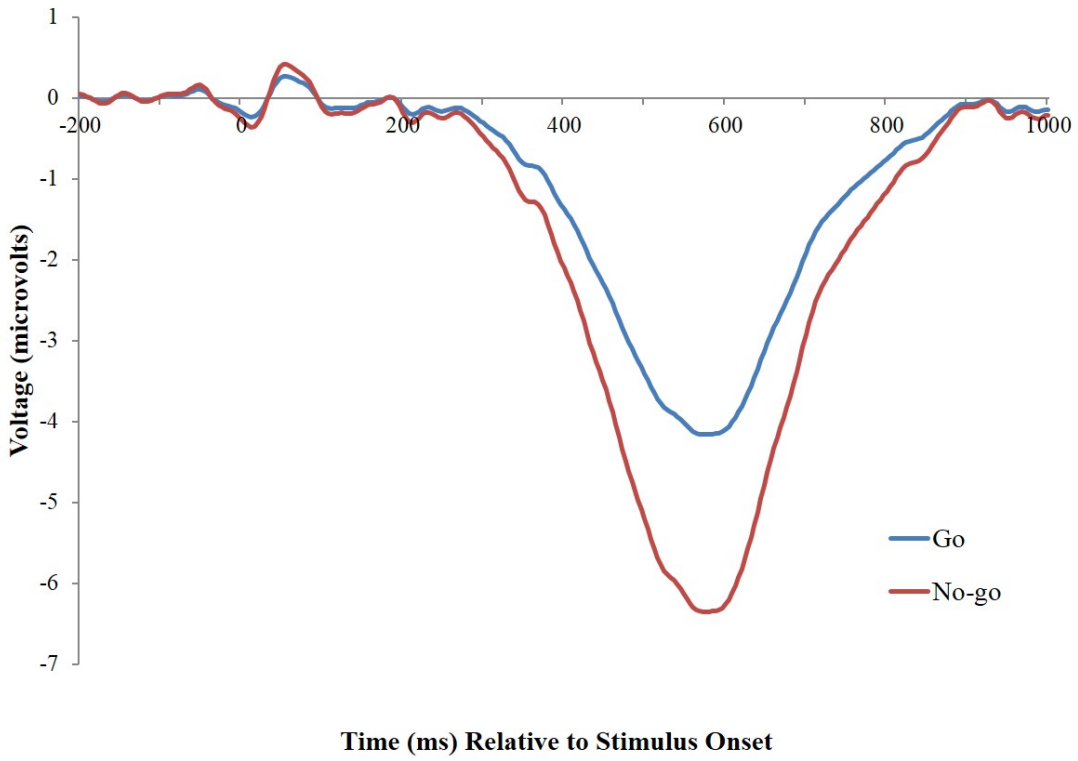

FIGURE 2.7: N2 ERP component in Bird/Alligator Task isolated by temporospatial PCA. Waveforms were averaged across electrodes from the frontocentral electrode cluster identified by the spatial PCA (see Figure 2.8).

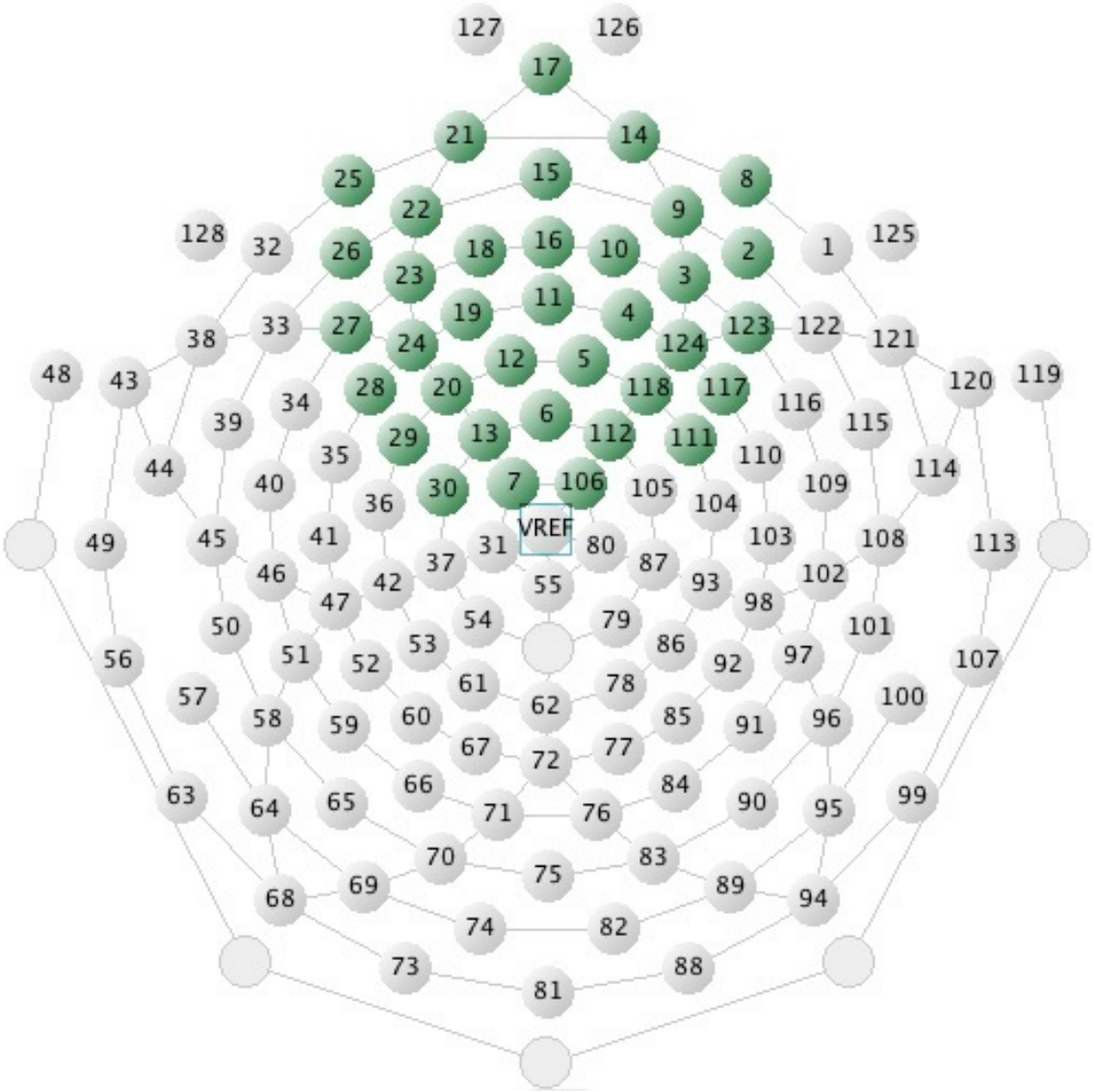

FIGURE 2.8: Electrode cluster for the frontocentral spatial component in the Bird/Alligator ERP task corresponding to the N2. Electrodes in green represent those electrodes with loadings of .40 or greater on the spatial PCA component.

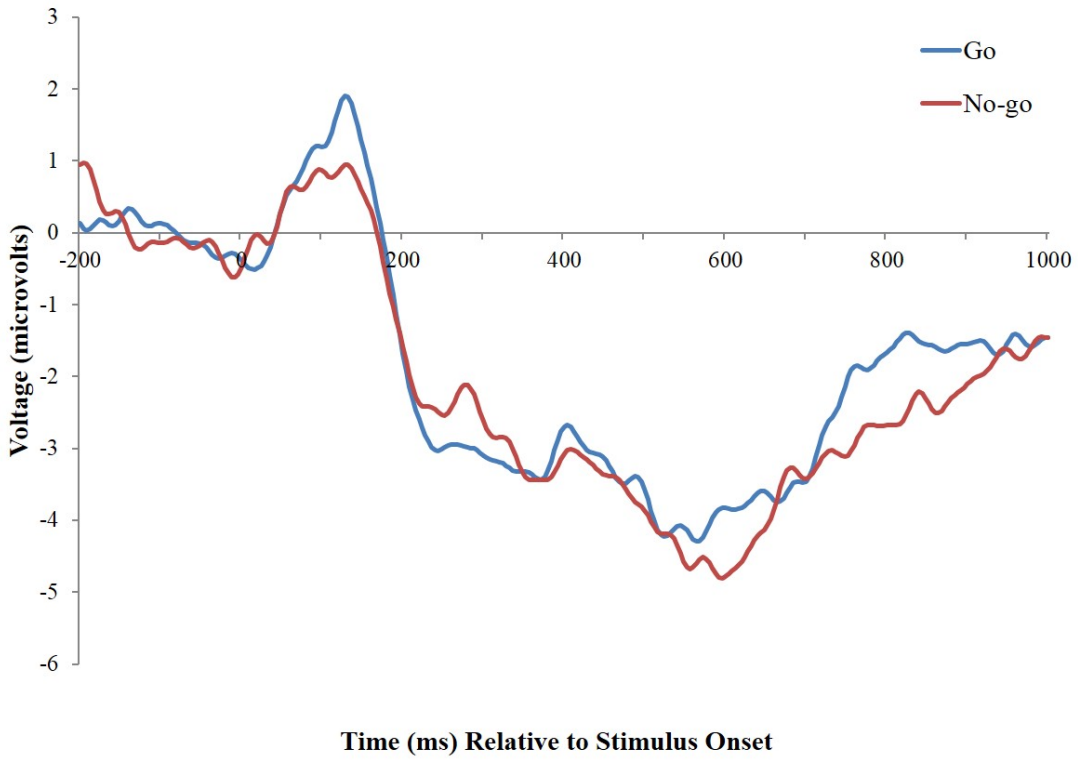

FIGURE 2.9: Frontocentral ERP waveform in Bird/Alligator task isolated by spatial PCA. Waveforms were averaged across electrodes from the frontocentral electrode cluster identified by the spatial PCA (see Figure 2.8).

N2 latencies, indicating no significant developmental changes in N2 amplitudes or latencies in the current sample.

A topo plot depicting the ERP waveforms grand-averaged across participants at each electrode site for the target and frequent conditions of the auditory oddball ERP task are in Figure 2.10. Grand-averaged ERP waveforms averaged across posterior electrodes for the target and frequent conditions are in Figure 2.11. A spatial PCA identified 13 spatial components accounting for 89% of the variance in the ERP waveform. A separate temporal PCA identified 14 temporal components accounting for 96% of the variance. A spatial PCA on the ERP data reduced by the temporal PCA (i.e., a temporo-spatial PCA) identified 5 spatial components accounting for 82% of the variance. The peak latency of the temporo-spatial component corresponding to the P3b was 716 ms (see Figure 2.12). The electrode cluster for the posterior spatial component corresponding to the P3b is displayed in Figure 2.13. The peak latency of the posterior spatial component corresponding to the P3b was 650 ms (see Figure 2.14). To identify each individual's peak latency of the P3 component, we identified each individual's latency to their peak maximum amplitude (because the P3b is a positive-going component) during the range of 650 ± 50 ms: 600–700 ms.

Descriptives of children's P3b amplitudes and latencies are in Table 2.1. Pearson correlations of children's P3b amplitudes and latencies are in Table 2.2. Unexpectedly, children's target P3b amplitudes were not significantly larger than their frequent P3b amplitudes ($t[21] = -0.18$, $p = .862$), calling into question whether the identified component reflects attentional processing of deviant stimuli. Among the 4 children with usable ERP data

TABLE 2.1: Study 1: Descriptive Statistics of Children's ERP Components.

	P3b					N2				
	Tgt Amp	Frq Amp	Amp Diff	Tgt Lat	Frq Lat	Go Amp	No-Go Amp	Amp Diff	Go Lat	No-Go Lat
N	22.00	22.00	22.00	21.00	22.00	34.00	34.00	34.00	33.00	33.00
M	8.11	8.63	−0.51	656.76	644.91	−6.48	−9.91	−3.42	569.58	580.85
SD	9.48	6.48	13.76	25.16	23.55	9.22	7.54	9.99	22.71	25.92
min	−16.92	−7.17	−27.08	612.00	608.00	−33.20	−35.18	−21.51	532.00	532.00
max	27.68	17.92	24.34	696.00	680.00	8.20	4.17	15.95	608.00	616.00

Note. "Amp" = amplitude, "Lat" = latencies, "Diff" = difference, "Tgt" = target, "Frq" = frequent. Amplitudes are in microvolts, latencies are in milliseconds. P3b amplitude difference reflects target P3b amplitude − frequent P3b amplitude. N2 amplitude difference reflects no-go N2 amplitude − go N2 amplitude.

TABLE 2.2: Study 1: Pearson Correlations of Children's ERP Components.

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.
1. Target P3b amplitude	–									
2. Frequent P3b amplitude	−.47*	–								
3. P3b amplitude difference	.91***	−.79***	–							
4. Target P3b latency	−.21	.15	−.22	–						
5. Frequent P3b latency	−.22	.04	−.17	.31	–					
6. Go N2 amplitude	.22	−.12	.21	.16	.30	–				
7. Nogo N2 amplitude	.42†	−.09	.33	.27	.09	.30†	–			
8. N2 amplitude difference	.12	.05	.06	.05	−.23	−.69***	.48**	–		
9. Go N2 latency	−.23	.23	−.27	.05	.23	−.05	.08	.10	–	
10. Nogo N2 latency	.06	−.06	.07	−.03	.13	−.04	−.15	−.08	−.04	–

Note. $^{\dagger}p < .10$. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$. Correlations are two-tailed.

on the oddball task at multiple assessments (out of 8 children with multiple EEG assessments), the cross-time continuity of the P3b amplitude was $r(2) = .76$ ($p = .244$) and P3b latency was $r(2) = .16$ ($p = .842$). Correlations between the P3b and child's age were $r(20) = .17$ ($p = .452$) for P3b amplitudes and $r(19) = .09$ ($p = .692$) for P3b latencies, suggesting there were no significant developmental changes in P3b amplitudes or latencies in the current sample.

2.1.2.1.4 EEG Data Processing. To examine the hypotheses of left frontal asymmetry (greater activation at left than right frontal electrodes) and less power in infant alpha (6–9 Hz) frequency bands in externalizing problems, we processed the data for EEG analysis. EEG data were analyzed during the segmented subject ERP averages (across trials) for the no-go and target conditions, for each task separately. We calculated alpha power using the fft function in MATLAB that calculates a discrete Fourier transform using a fast Fourier transform algorithm to decompose the EEG signal into its composing frequencies. For calculating frontal alpha power, we selected the frontal electrode cluster based on a spatial PCA, which resulted in the frontocentral spatial component corresponding to the N2 (see Figure 2.8). For calculating frontal asymmetry, we selected left frontal (see Figure 2.15) and right frontal (see Figure 2.16) electrodes based on *a priori* regions of interest. Log-transformed power values across frequencies at frontal electrodes in each task are depicted in Figure 2.17. We examined alpha power in the 6–9 Hz frequency band because 6–9 Hz power has been shown to be related to children's self-regulation (e.g., Wolfe & Bell, 2004), it is the dominant frequency band in 2–3-year-olds (Marshall, Bar-Haim, & Fox, 2002), and it is widely examined in the literature. We computed the average power at these electrode clusters in the 6–9 Hz frequency band, and normalized the power values with

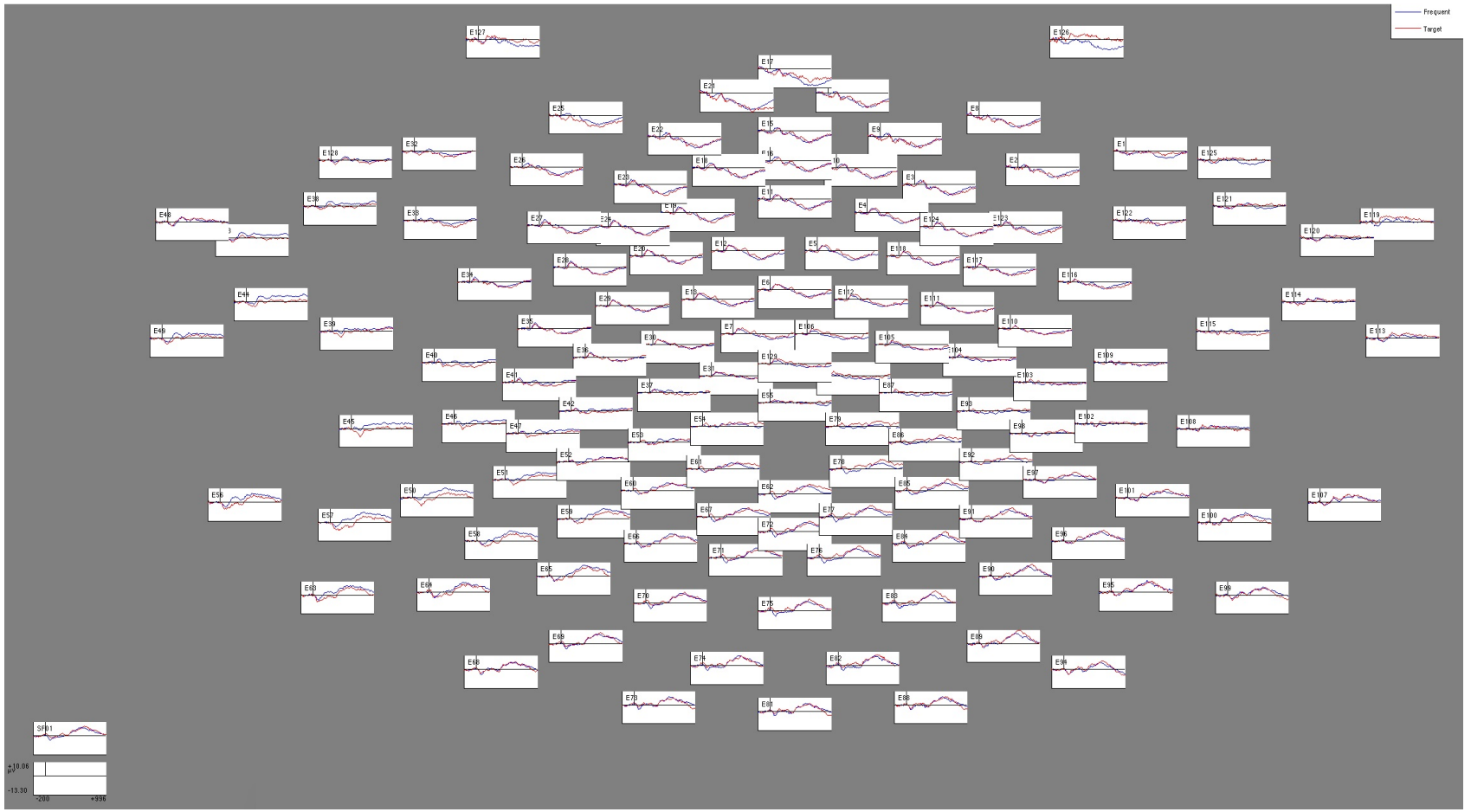

FIGURE 2.10: Topo plot of grand-averaged ERP waveforms for the target (red) and frequent (blue) conditions of the auditory oddball task. The positive-going wave corresponding to the P3b can be observed in the posterior electrodes. Frontal electrodes (e.g., E17) are located at top of diagram, posterior electrodes (e.g., E81) at bottom.

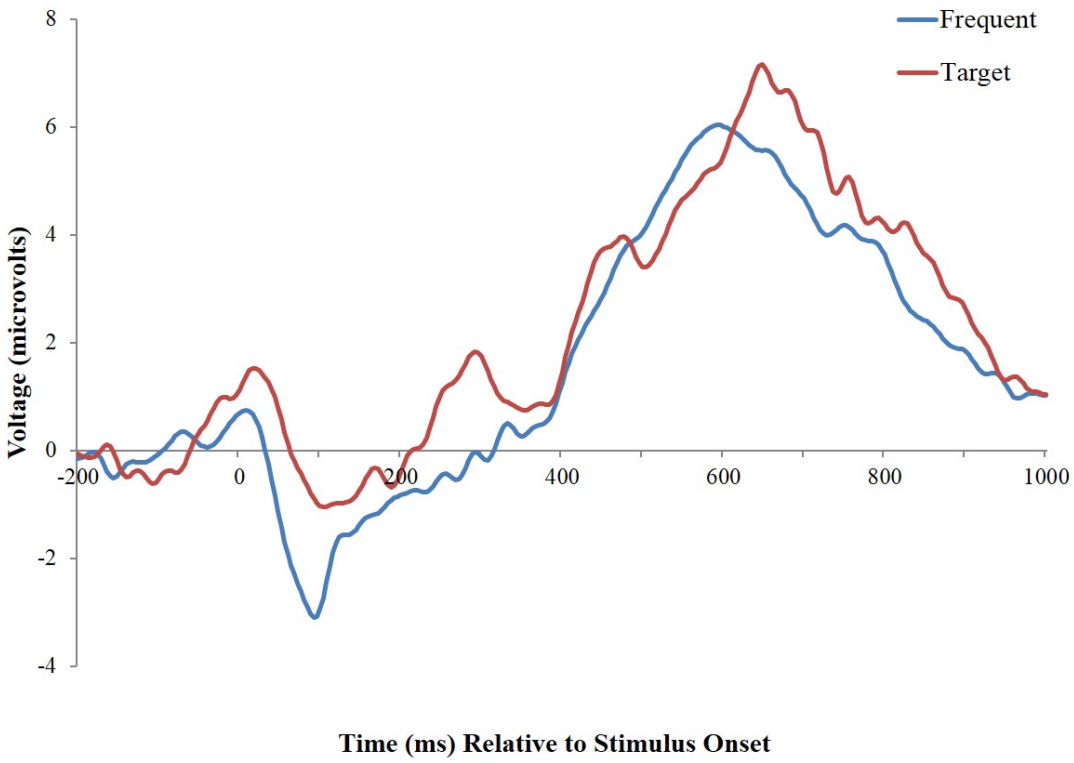

FIGURE 2.11: Grand-averaged ERP waveforms averaged across posterior electrodes for the target and frequent conditions of the oddball task. Waveforms were averaged across electrodes from the posterior electrode cluster identified by the spatial PCA (see Figure 2.13).

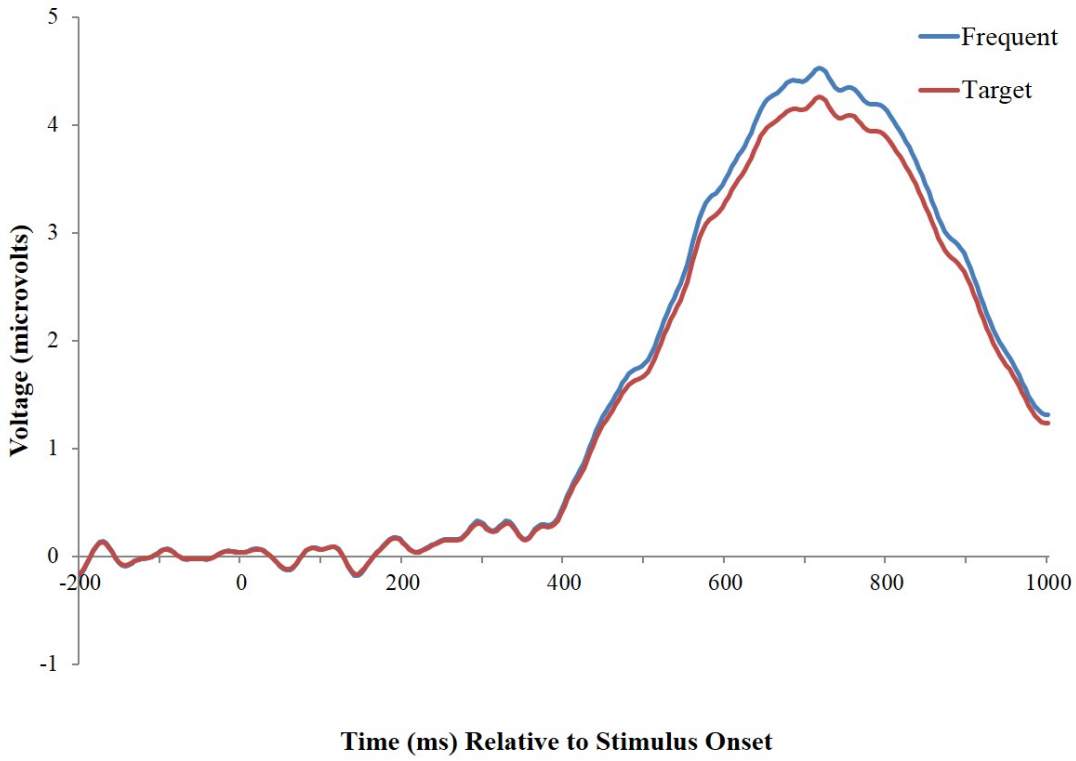

FIGURE 2.12: P3b ERP component in oddball task isolated by temporospatial PCA. Waveforms were averaged across electrodes from the posterior electrode cluster identified by the spatial PCA (see Figure 2.13).

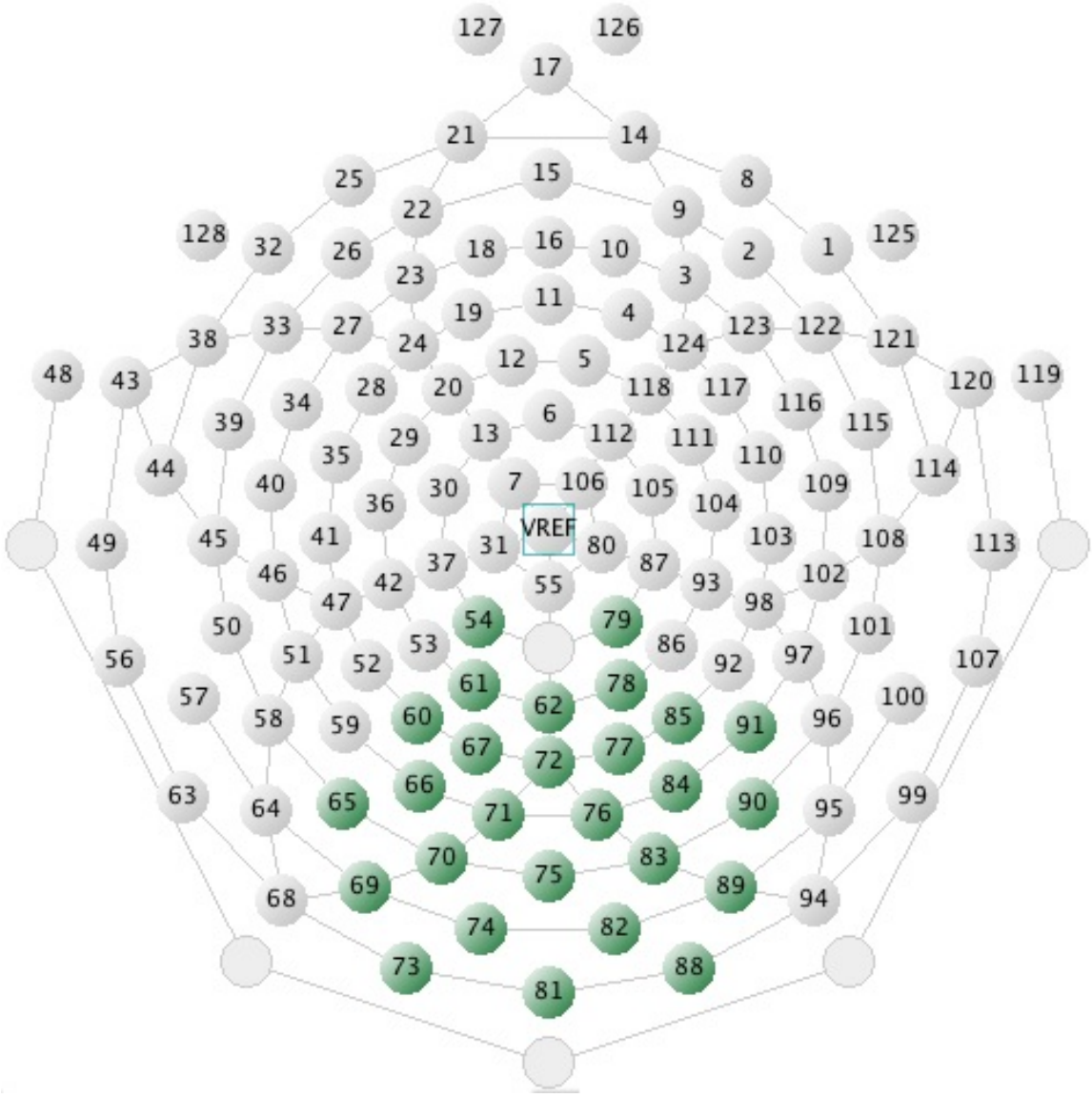

FIGURE 2.13: Electrode cluster for the posterior spatial component in the oddball task corresponding to the P3b. Electrodes in green represent those electrodes with loadings of .40 or greater on the spatial PCA component.

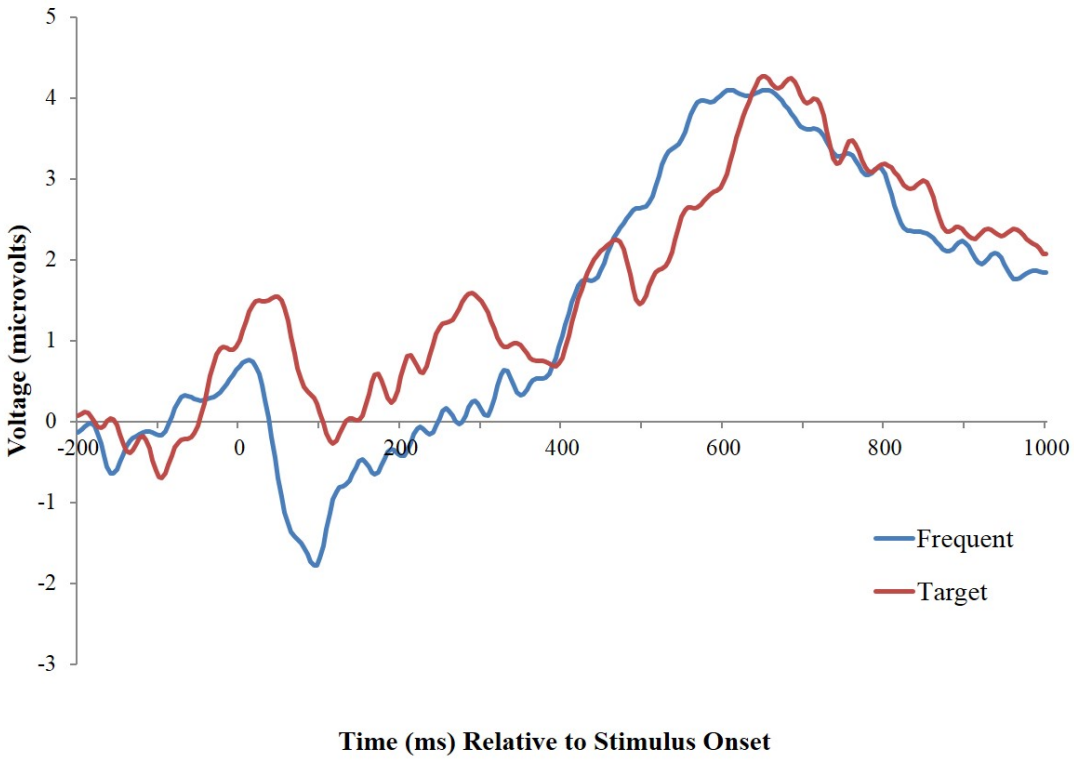

FIGURE 2.14: Posterior ERP waveform in oddball task isolated by spatial PCA. Waveforms were averaged across electrodes from the posterior electrode cluster identified by the spatial PCA (see Figure 2.13).

a log transformation. Frontal EEG asymmetry scores were calculated by subtracting left frontal alpha power from right frontal alpha power (i.e., asymmetry score = right frontal alpha power − left frontal alpha power). Higher alpha power is inversely associated with cortical activation (Fox, 1994), so higher alpha power values at right than left electrodes reflects greater cortical activation at left than right electrodes. Thus, positive asymmetry scores reflect left frontal asymmetry (greater left than right activation) and negative scores reflect right frontal asymmetry. To examine frontal alpha power, we used the frontal region of electrodes selected by the spatial PCA (see Figure 2.8).

Descriptives of children's frontal power and frontal asymmetry scores are in Table 2.3. Pearson correlations of children's frontal power and frontal asymmetry scores are in Table 2.4. Frontal alpha power was positively associated across the oddball and Bird/Alligator tasks. Frontal asymmetry was marginally positively associated across the tasks. This is consistent with findings of greater reliability of frontal alpha power than frontal asymmetry in children (Vuga et al., 2008). A power spectrum decomposition showed similar power (amplitude in squared microvolts) across the oddball and Bird/Alligator tasks, with higher power at low than at high frequencies. For the power spectrum decomposition, see Figure 2.17.[1]

We saw some (non-significant) evidence of cross-time continuity in our very small sample. Among the 8 children with multiple EEG assessments, the cross-time continuity of frontal alpha power was $r(2) = .67$ ($p = .328$) in the oddball task and $r(5) = .46$ ($p = .296$) in the Bird/Alligator task. The cross-time continuity of frontal asymmetry was $r(2) = -.73$ ($p = .272$) in the oddball task and $r(5) = .46$ ($p = .296$) in the Bird/Alligator task.

[1]The low-pass filter of 30 Hz attenuates but does not fully remove frequencies above 30 Hz.

Correlations between frontal alpha power and child's age were $r(20) = .21$ ($p = .360$) in the oddball task and $r(32) = .01$ ($p = .968$) in the Bird/Alligator task. Correlations between left frontal asymmetry and child's age were $r(20) = -.08$ ($p = .739$) in the oddball task and $r(32) = -.11$ ($p = .520$) in the Bird/Alligator task. This suggests there were no significant developmental changes in frontal alpha power or frontal asymmetry in the current sample.

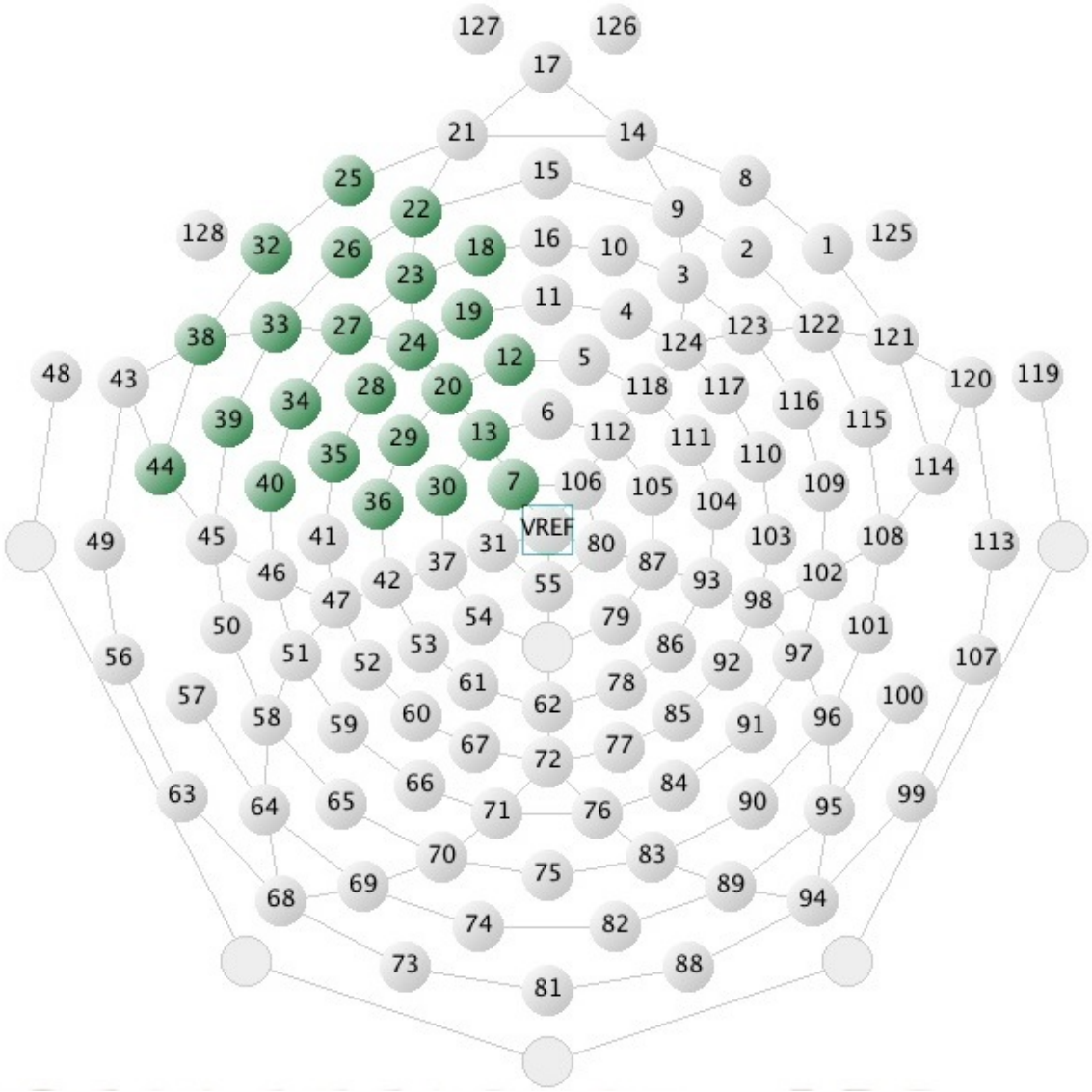

FIGURE 2.15: Diagram of left frontal electrodes (in green).

TABLE 2.3: Study 1: Descriptive Statistics of Children's EEG and Time-Frequency Components.

	Oddball			Bird/Alligator		
	Frontal Power	Frontal Asymmetry	Frontal TF	Frontal Power	Frontal Asymmetry	Frontal TF
N	22.00	22.00	22.00	34.00	34.00	34.00
M	4.00	−0.04	0.47	3.72	−0.07	0.09
SD	0.49	0.39	4.46	0.46	0.37	3.52
min	2.81	−0.83	−7.35	2.63	−0.89	−7.71
max	4.68	0.72	10.89	4.58	0.73	8.12

Note. "TF" = time-frequency activity corresponding to timing of P3b (oddball) or N2 (Bird/Alligator), with values in decibels. Power values were log-transformed. Frontal power and asymmetry in alpha frequency range. Frontal time-frequency in theta frequency range. Frontal alpha asymmetry reflects right frontal alpha power − left frontal alpha power.

Table 2.4: Study 1: Pearson Correlations of Children's EEG and Time-Frequency Components.

	1.	2.	3.	4.	5.	6.
1. Oddball Frontal Alpha Power	–					
2. Oddball Frontal Alpha Asymmetry	.20	–				
3. Oddball P3b-Related Frontal Theta	−.08	.14	–			
4. Bird/Alligator Frontal Alpha Power	.67**	.23	−.22	–		
5. Bird/Alligator Frontal Alpha Asymmetry	.08	.40†	.34	.03	–	
6. Bird/Alligator N2-Related Frontal Theta	−.05	.16	.06	−.06	.12	–

Note. $^{\dagger}p < .10$. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$. Correlations are two-tailed.

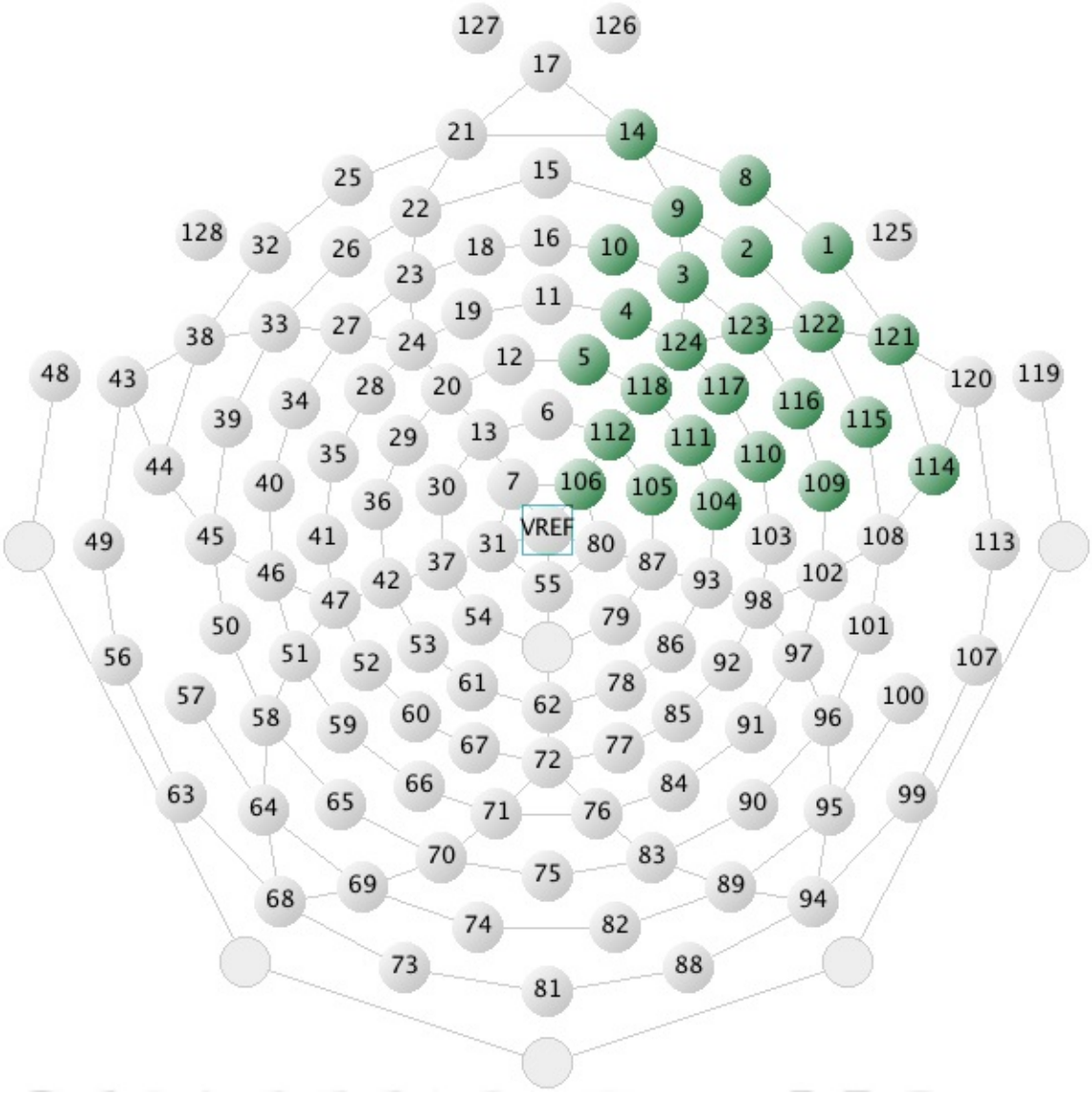

FIGURE 2.16: Diagram of right frontal electrodes (in green).

2.1.2.1.5 Time-Frequency Data Processing. To examine the hypothesis of less frontal theta power during the timing of the N2 and P3 in children with externalizing problems, we processed the data for time-frequency analysis. EEG data were analyzed during the segmented ERP trials for each task separately. Based on prior findings, we were particularly interested in the theta power linked to the no-go N2 and oddball target P3 ERPs, so we

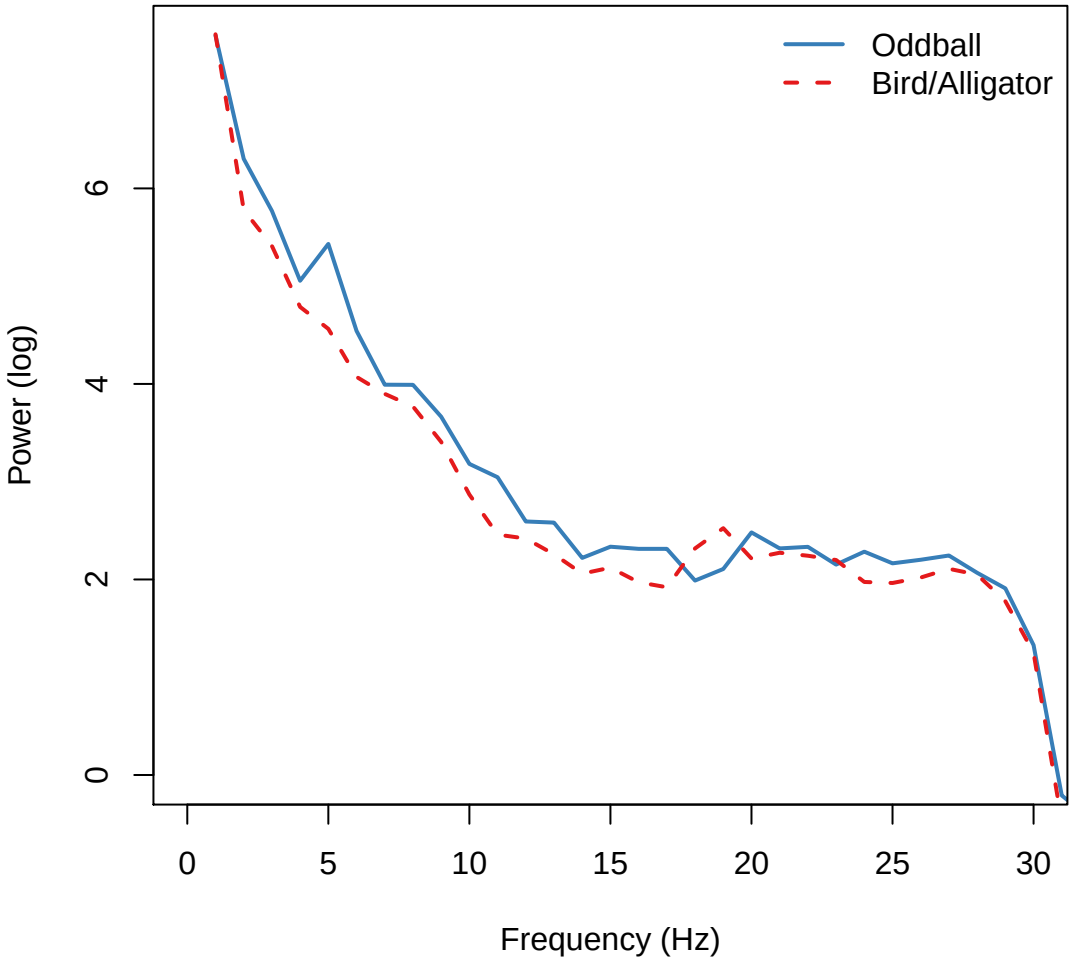

FIGURE 2.17: Power spectrum decomposition of the EEG waveforms. Log-transformed power values across frequencies at frontal electrodes (see Figure 2.8) in the Bird/Alligator and oddball tasks.

examined the EEG data in the no-go and oddball target conditions only. For time-frequency analysis, we examined frontal theta activity (4–5 Hz) from the decomposition of frequencies at the time frames corresponding to the no-go N2 (524–624 ms) and oddball P3 (600–700 ms). Even though the oddball P3 typically has a parietal spatial distribution, we examined frontal theta activity because of prior findings relating P3-related frontal theta activity to externalizing problems such as alcoholism (Jones et al., 2006; Porjesz, Rangaswamy, et al., 2005). We used the frontal region of electrodes selected by the spatial PCA (see Figure 2.8). We conducted the time-frequency analysis in the EEGLAB (Delorme & Makeig, 2004) toolbox (version 13.4.4b) for MATLAB. Time-frequency analysis in EEGLAB convolved the ERP waveform with a Morlet wavelet to measure the amount of activation in each successive, overlapping time window with a 1.2-cycle (oscillations per second in Hz) wavelet and a Hanning-tapered window. The number of cycles in the wavelet used for higher frequencies expanded slowly over the time course of the waveform, reaching half the number of cycles in the equivalent fast Fourier transform window at its highest frequency. Units of the variables derived from the time-frequency analysis are in decibels, which are defined as:

$$\mathrm{dB} = \frac{10 \times \mathrm{Log(Power)}}{\mathrm{Hz}} \tag{2.1}$$

where Power is the voltage of the EEG signal in squared microvolts. Note that decibels are referenced relative to the baseline period, so they can be negative. Time-frequency analysis plots are in Figures 2.18 and 2.19 for the Bird/Alligator and oddball tasks, respectively.

Descriptive statistics of children's time-frequency components are in Table 2.3. Pearson correlations of children's time-frequency components are in Table 2.4. Among the 8 children with multiple EEG assessments, there was no evidence of cross-time continuity. The cross-time continuity of P3b-related frontal theta activity in the oddball task was $r(2) = -.53$ ($p =$

.472). The cross-time continuity of N2-related frontal theta activity in the Bird/Alligator task was $r(5) = .15$ ($p = .745$). The correlation between P3b-related frontal theta activity and child's age in the oddball task was $r(20) = .11$ ($p = .622$). The correlation between P3b-related frontal theta activity and child's age in the oddball task was $r(32) = -.18$ ($p = .299$), suggesting no significant developmental changes in these components in the current sample.

2.1.2.2 Self-Regulation

In addition to ERP tasks, the protocol included multitrait-multimethod measures of constructs from the leading models of development of self-regulation. To assess risk for externalizing psychopathology, behavioral tasks were chosen that tap into multiple domains of self-regulation, in which deficits are related to externalizing psychopathology. a) Inhibitory control: Three tasks measured the child's ability to inhibit responses to irrelevant, prepotent stimuli, similar to impulse control. b) Sustained attention: Two tasks measured the child's ability to maintain attentional focus to relatively uninteresting stimuli. Only a later part of the sample (17 children, 63%) was given the opportunity to complete the sustained attention tasks. Therefore, analyses involving the sustained attention tasks only included these 17 children. Descriptive statistics (means, standard deviations, minima, and maxima) of the self-regulation tasks are presented in Table 2.5. The correlations among the self-regulation tasks are in Table 2.6. The correlations among the self-regulation tasks were modest (with most rs ranging from .10 to .30).

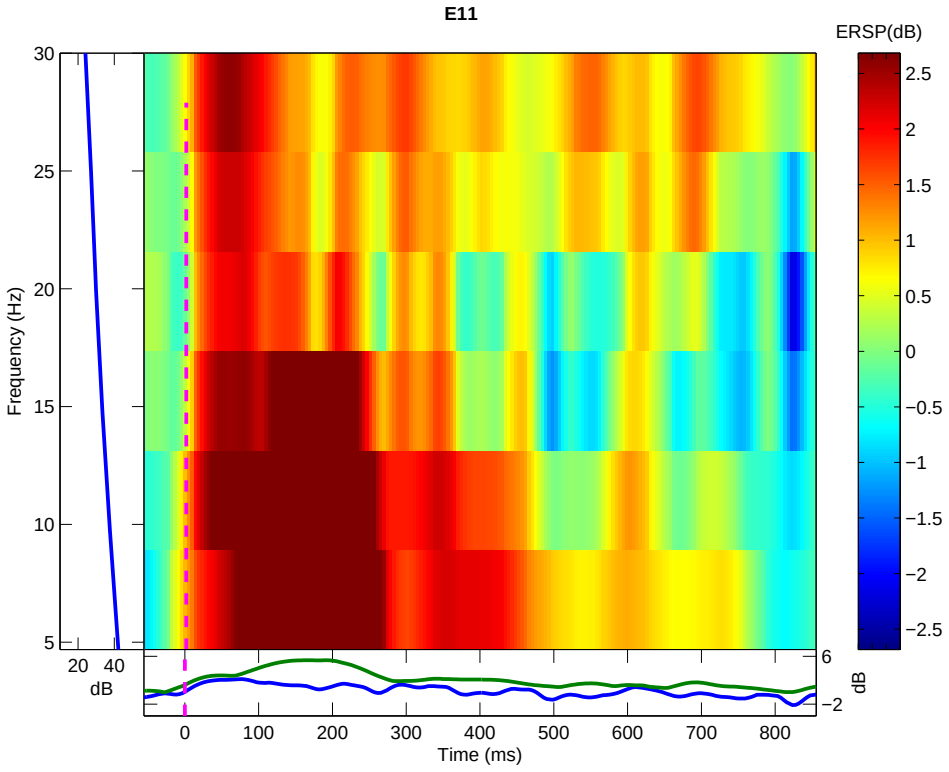

FIGURE 2.18: Time-frequency analysis plot of event-related spectral perturbation values (in decibels) from a frontal electrode (E11) in the Bird/Alligator task. Actual time-frequency estimates were averaged across electrodes from the frontal electrode cluster identified by the spatial PCA (see Figure 2.8). Frontal N2-related theta activity corresponds to high power (red shading) from 524 to 624 ms in the 4–5 Hz frequency band.

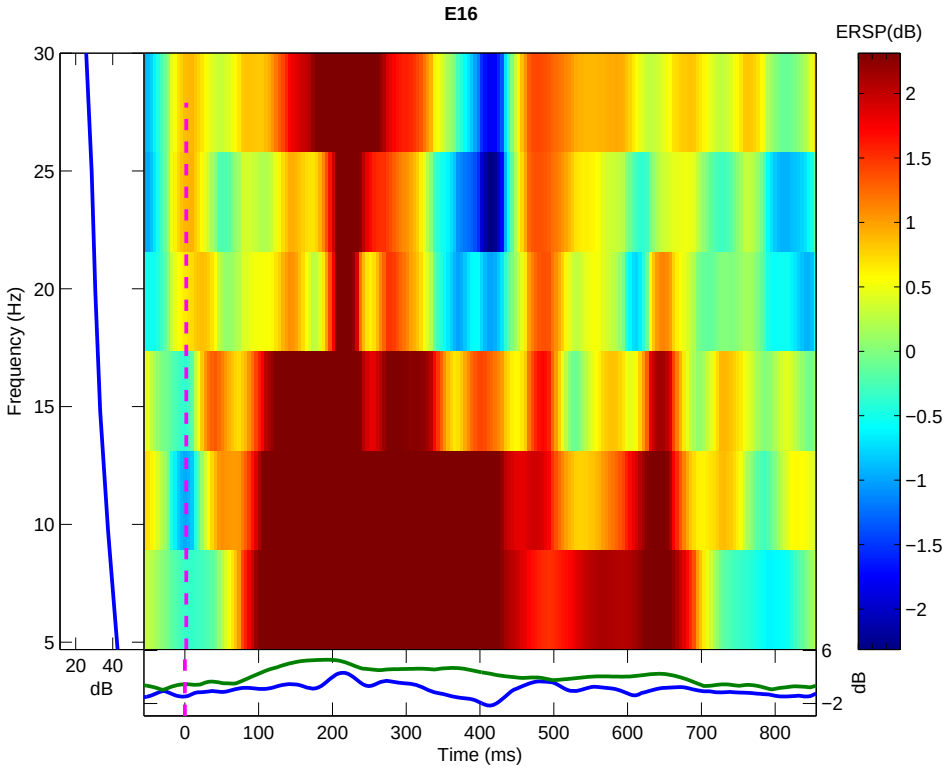

FIGURE 2.19: Time-frequency analysis plot of event-related spectral perturbation values (in decibels) from a frontal electrode (E16) in the oddball task. Actual time-frequency estimates were averaged across electrodes from the frontal electrode cluster identified by the spatial PCA (see Figure 2.8). Frontal P3b-related theta activity corresponds to high power (red shading) from 600 to 700 ms in the 4–5 Hz frequency band.

TABLE 2.5: All Studies: Descriptive Statistics of the Self-Regulation Tasks.

	Bird/Alligator			Shape Stroop			Grass/Snow			Token Sort			Sustained Play Attention		
Age (mo)	30	36	42	30	36	42	30	36	42	30	36	42	30	36	42
N	172.00	151.00	154.00	213.00	168.00	156.00	142.00	134.00	122.00	193.00	141.00	123.00	189.00	125.00	105.00
M	0.84	1.35	2.17	0.99	1.52	1.75	0.35	0.44	0.64	37.77	65.06	94.63	0.10	0.11	0.10
SD	0.93	1.19	1.13	0.70	0.59	0.43	0.26	0.32	0.31	42.17	59.64	61.96	0.09	0.10	0.10
min	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
max	3.00	3.00	3.00	2.00	2.00	2.00	1.00	1.00	1.00	180.00	180.00	180.00	0.54	0.50	0.59

Note. "mo" = months. "All Studies" refers to the full sample of 336 families who were part of the larger study (i.e., not just those who were recruited for the EEG procedures). "Bird/Alligator" refers to the behavioral inhibitory control task, not the Bird/Alligator ERP task.

2.1.2.2.1 Inhibitory Control Tasks. Inhibitory control was measured by three different behavioral tasks: Bird/Alligator, Grass/Snow, and Shape Stroop. These tasks were chosen because they (or similar variants) are widely used and are thought to reflect important aspects of self-regulation, and because they are useful for measuring normative individual differences in inhibitory control across the target age range of 30 to 42 months (Petersen, Hoyniak, McQuillan, & Bates, under review). Garon, Bryson, and Smith (2008) described these or similar tasks as measures of complex response inhibition, in which the child has to (1) hold a rule in mind, (2) respond according to the rule, and (3) inhibit a prepotent response. It is worth noting that although we refer to these as measures of inhibitory control, inhibitory control and self-regulation tasks (and cognitive tasks in general) are multidimensional and reflect other processes including working memory (Wolfe & Bell, 2007).

Bird/Alligator (adapted from Kochanska, Murray, Jacques, Koenig, & Vandegeest, 1996; Reed, Pien, & Rothbart, 1984) is a Simon-says task in which the child has to follow the directions given by the bird puppet, but to ignore commands from the alligator (see Figure 2.20). The children played several practice trials and then were presented with 12 trials, including six go (i.e., bird) trials and six no-go (i.e., alligator) trials. After six trials, the

Table 2.6: All Studies: Pearson Correlations of the Self-Regulation Tasks.

Task		Bird/Alligator			Shape Stroop			Grass/Snow			Token Sort			Sustained Play Attention		
	Age (mo)	30	36	42	30	36	42	30	36	42	30	36	42	30	36	42
Bird/Alligator	30	–														
Bird/Alligator	36	.19*	–													
Bird/Alligator	42	.02	.38***	–												
Shape Stroop	30	.00	.15†	.15	–											
Shape Stroop	36	−.02	.14†	.24*	.10	–										
Shape Stroop	42	−.07	.08	.18*	.28**	.28**	–									
Grass/Snow	30	.17†	.20†	−.06	−.10	−.23*	−.07	–								
Grass/Snow	36	.09	.26**	.05	.07	.00	−.07	.11	–							
Grass/Snow	42	.16	.18†	.10	.07	.17†	.15	−.04	.24*	–						
Token Sort	30	−.06	.03	.07	−.02	−.10	.07	.09	−.10	.07	–					
Token Sort	36	.04	.09	.15	−.04	.12	.21*	.27**	.03	.12	.29**	–				
Token Sort	42	−.10	−.12	.30**	−.08	.22*	.24**	−.01	−.02	.20*	.20*	.46***	–			
Sustained Play Attention	30	.13	−.06	.02	−.06	.22*	.14	−.08	.12	.13	−.01	−.03	.16†	–		
Sustained Play Attention	36	.23*	.00	−.05	−.05	.11	−.20*	.00	.01	.06	−.04	−.13	−.06	.25**	–	
Sustained Play Attention	42	.09	.09	.00	.00	.09	.07	−.02	−.06	.01	.01	.07	.08	.08	.11	–

Note. $^{\dagger}p < .10$. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$. "mo" = months. "All Studies" refers to the full sample of 336 families who were part of the larger study (i.e., not just those who were recruited for the EEG procedures). "Bird/Alligator" refers to the behavioral self-regulation task, not the Bird/Alligator ERP task. Correlations are two-tailed. *N*s range from 68 to 181.

participants received a reminder of the rules. If participants successfully demonstrated action on the go trials and inhibition on the no-go trials at 36 months, an additional 12 trials were presented with a rule-switch in which the alligator trials were go trials and the bird trials were no-go trials. At 42 months, all children received the rule-switch. Each no-go trial was scored on a 0 to 3 scale (0 = full commanded movement, i.e., full mistake, 1 = partial movement, 2 = wrong movement, and 3 = no movement) according to the scoring system used by Carlson and Moses (2001). The final Bird/Alligator score was the child's average score on no-go trials (0–3). The interrater reliability for Bird/Alligator, based on intraclass correlation (ICC[2, $k = 14$]), was .98. Children who had scores for Bird/Alligator numbered 19 (70%) at 30 months, 18 (67%) at 36 months, and 14 (52%) at 42 months. Much of the missingness at the later ages owed to participants not having yet aged into the target age, and thus were not yet eligible for assessment. Cross-time continuity of Bird/Alligator scores was $r(104) = .19$ ($p = .049$) from 30 to 36 months and $r(108) = .38$ ($p < .001$) from 36 to 42 months. Cross-time continuity of Bird/Alligator scores did not show significant increases over time from 30–36 to 36–42 months ($t = -1.45$, $p = .151$).

In *Shape Stroop* (Kochanska, Murray, & Harlan, 2000), the child has to point to pictures of small fruit embedded within pictures of different, larger fruit (see Figure 2.21). The child was presented with three pictures, in which each contained a small fruit in the middle of a larger fruit. In three of the trials, the child was asked to point to a large fruit out of the set (e.g., the large banana). After the three large fruit trials, the child was asked to point to a small fruit out of the set (e.g., the small apple) in three more trials. Each small fruit trial was scored from 0 to 2 (0 = incorrect, 1 = initially incorrect, but changed response to correct, 2 = correct). The final Shape Stroop score was the average score on the small fruit

FIGURE 2.20: Picture of puppets in Bird/Alligator inhibitory control task.

trials (0–2). Interrater reliability for Shape Stroop was ICC(2, $k = 54$) = .99. Children who had scores for Shape Stroop numbered 21 (78%) at 30 months, 19 (70%) at 36 months, and 14 (52%) at 42 months. Cross-time continuity of Shape Stroop scores was $r(146) = .10$ ($p = .227$) from 30 to 36 months and $r(119) = .28$ ($p = .002$) from 36 to 42 months. Cross-time continuity of Shape Stroop scores showed marginally significant increases over time from 30–36 to 36–42 months ($t = -1.86$, $p = .065$).

In *Grass/Snow* (Carlson & Moses, 2001), the child has to touch a white square when the experimenter says "Grass" and a green square when hearing "Snow" (see Figure 2.22). The child was given several practice trials and was then presented with 12 trials, six of each color, and each trial was scored either correct (1) or incorrect (0). The final score represented the sum of all correct responses (0–12). Interrater reliability for Grass/Snow

FIGURE 2.21: Picture of fruit stimuli used in Shape Stroop task.

was ICC(2, $k = 52$) = .98. Children who had scores for Grass/Snow numbered 19 (70%) at 30 months, 19 (70%) at 36 months, and 13 (48%) at 42 months. Cross-time continuity of Grass/Snow scores was $r(95) = .11$ ($p = .290$) from 30 to 36 months and $r(92) = .24$ ($p = .021$) from 36 to 42 months. Cross-time continuity of Grass/Snow scores did not show significant increases over time from 30–36 to 36–42 months ($t = -0.89$, $p = .373$).

2.1.2.2.2 Sustained Attention Tasks. *Token Sort* is a task modified from a bead sorting task (Goldsmith, Reilly, Lemery, Longley, & Prescott, 1999) designed to assess how well children sustain attention in a low stimulation, academic-like task. In front of the child, the experimenter placed a container with tokens of three colors (red, white, and blue) intermixed (see Figure 2.23). Then the experimenter asked the child to sort the tokens by color into three smaller containers (see Figure 2.23). After giving the child instructions and demonstrating putting one of each color token in its respective bin, the experimenter left the room. The task began when the experimenter left the room and lasted 3 minutes. The total time the child sorts the tokens was used as a measure of sustained attention.

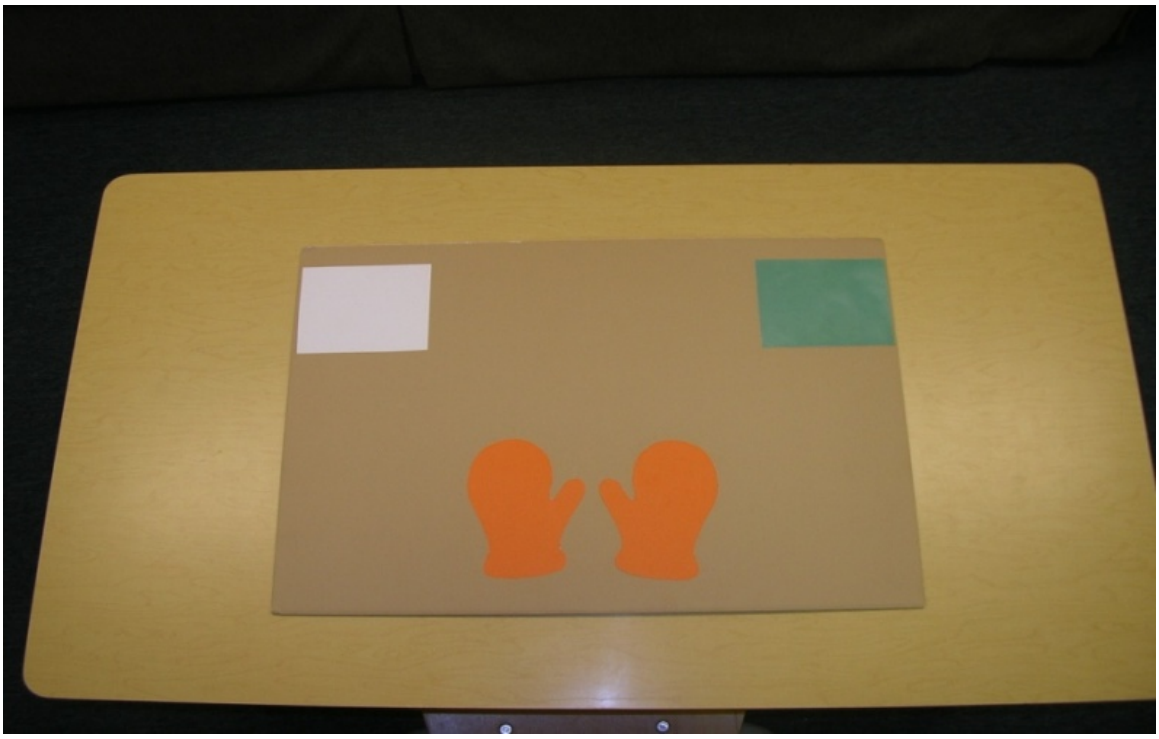

FIGURE 2.22: Picture of Grass/Snow task.

Interrater reliability for the Token Sort task was ICC(2, $k = 6$) = 1.00. Children who had scores for the Token Sort task numbered 17 (100%) at 30 months, 13 (76%) at 36 months, and 13 (76%) at 42 months. Cross-time continuity of Token Sort scores was $r(126) = .29$ ($p = .001$) from 30 to 36 months and $r(104) = .46$ ($p < .001$) from 36 to 42 months. Cross-time continuity of Token Sort scores showed marginally significant increases over time from 30–36 to 36–42 months ($t = -1.75$, $p = .082$).

Sustained Play Attention is a task in which the child is asked to play alone with relatively uninteresting toys at a table (Ruff, Capozzoli, & Weissberg, 1998). Next to the table with the ordinary toys (see Figure 2.24), however, was a cart with more attractive toys (see Figure 2.25), with which the child had been instructed not to play. The experimenter instructed the child to stay seated at the table and to play with the toys while

FIGURE 2.23: Picture of Token Sort task.

the experimenter and the child's mother do some work. The mother was seated behind a partition, but was accessible to the child if he or she chose. The experimenter stayed seated across from the child while quietly working on paperwork. The task lasted 5 minutes. Child attention during the task was coded second by second with three levels: casual, settled, and focused. Casual attention was defined as "looking at the toys but not being engaged. For the older children, casual attention was usually manifested by looking around the display and picking up toys and putting them back down again, that is, searching the array of toys for something to do." (Ruff & Capozzoli, 2003, p. 879). Settled attention was defined as "a pause in the child's casual attention to look at and manipulate a particular toy [i.e., from a set of toys within a free-play period]. Looking was steady but not necessarily intent, extraneous movement tended to diminish but might have been present, and there might have been some talking." (Ruff & Capozzoli, 2003, p. 879). Focused attention was defined

as "concentrated attention that involved an intent facial expression, minimal extraneous bodily activity, a posture that enclosed the object of interest and brought it closer to eyes, and either no talking or soft talking clearly directed to the self" (relevant to the toys the child is playing with; Ruff & Capozzoli, 2003, p. 879). For an attention level to be coded, the behavior had to exceed 500 ms in duration. We first calculated the amount of time the child spent in each of the three levels of attention. We then calculated the proportion of time for each attention level out of the total duration of the task. A proportion score was used because the task did not last 5 minutes for some children ($n = 6$) due to experimenter error or another reason (e.g., the child became distressed before the end of the task, so we ended the task before 5 minutes). We used the proportion of time the child spent in focused attention as our measure of sustained attention. Higher values on the proportion score reflected a greater degree of sustained attention during the task.

Interrater reliability for the Sustained Play Attention task was ICC(2, $k = 13$) = .77. Children who had scores for the Sustained Play Attention task numbered 17 (100%) at 30 months, 13 (76%) at 36 months, and 10 (59%) at 42 months. Cross-time continuity of Sustained Play Attention scores was $r(116) = .25$ ($p = .007$) from 30 to 36 months and $r(92) = .11$ ($p = .305$) from 36 to 42 months. Cross-time continuity of Sustained Play Attention scores did not show significant increases over time from 30–36 to 36–42 months ($t = 0.27$, $p = .790$).

The low-to-moderate cross-time correlations of the self-regulation measures suggests that (a) self-regulation development is in flux during toddlerhood (i.e., knowing how well a child is able to regulate his or her behavior at 30 months of age may not be especially informative for knowing how well the child will be able to self-regulate 6 months later) and/or (b)

these tasks are not very reliable for measuring self-regulation in young children. It will be important in the future to evaluate possible developmental change versus lack of reliability by (a) testing shorter intervals for test-retest, and (b) by improving the representation of tasks for different developmental stages. The low-to-moderate inter-task correlations suggests that the tasks may be tapping somewhat different self-regulatory processes from each other.

FIGURE 2.24: Picture of relatively uninteresting toys at the table during the Sustained Play Attention task. Next to the table with the ordinary toys was a cart with more attractive toys (see Figure 2.25), with which the child had been instructed not to play. The child was instructed to stay seated at the table and play with the toys independently.

2.1.2.3 Externalizing Behavior Problems

Questionnaires were completed by a parent and secondary caregiver to provide multiple perspectives of the child's behavior. 1. *Achenbach Child Behavior Checklist 1 ½–5* (CBCL; Achenbach & Rescorla, 2000): includes two primary factors of externalizing behavior, attention problems and aggressive behavior, that compose a factor of externalizing problems,

FIGURE 2.25: Picture of attractive toys on the cart, situated 1 meter to the child's left (90°to the child's task table with relatively uninteresting toys, see Figure 2.24). During the Sustained Play Attention task, the child was instructed to stay seated at the table and play with the toys independently.

which represents the sum of 24 items such as, "defiant," "attacks people," and "can't sit still." Reporters rated whether, over the past 2 months, a given behavior was "not true," "somewhat or sometimes true," or "very true or often true" (scored 0, 1, and 2, respectively). The Achenbach scales are among the best normed and most widely used measures for behavior problems in this age range. They have good test-retest reliability and satisfactory content, criterion, and construct validity (Sattler & Hoge, 2006). Although the Attention Problems subscale is not a diagnostic checklist of ADHD symptoms, it has been interpreted as a measure of ADHD symptoms because it assesses the three dimensions of ADHD symptoms: inattention, hyperactivity, and impulsivity (Lifford, Harold, & Thapar, 2008). It is associated with other measures of ADHD, including the Conners rating scale (Conners, 1969) and DSM-IV symptoms of ADHD (American Psychiatric Association, 2000; Derks, Hudziak, Dolan, van Beijsterveldt, Verhulst, & Boomsma, 2008). In addition, it has been shown to measure ADHD as well as the Conners Scale does (Derks et al., 2008), with strong sensitivity and specificity (Chen, Faraone, Biederman, & Tsuang, 1994). 2. *Eyberg Child Behavior Inventory* (ECBI; Eyberg & Pincus, 1999): includes an index of intensity of behavior problems that is sensitive to change in child behavior. The index includes 36 behaviors including "has temper tantrums," "refuses to obey until threatened with punishment," and "has short attention span." Parents reported how often the behaviors currently occur on a 1 to 7 scale, where 1 is "never" and 7 is "always." The scores are summed across items to create an index of intensity of behavior problems.

Children who had parent reports on the CBCL numbered 23 (85%) at 30 months, 23 (85%) at 36 months, and 12 (44%) at 42 months. Children who had secondary caregiver reports on the CBCL numbered 3 (11%) at 30 months, 7 (26%) at 36 months, and 7

(26%) at 42 months. Children who had parent reports on the ECBI numbered 17 (63%) at 30 months, 16 (59%) at 36 months, and 13 (48%) at 42 months. Externalizing problem ratings showed relatively high cross-time continuity (with most rs ranging from .40 to .60). Cross-time continuity of parents' reports of externalizing problems on the CBCL was $r(168) = .59$ ($p < .001$) from 30 to 36 months and $r(134) = .75$ ($p < .001$) from 36 to 42 months. Cross-time continuity of secondary caregivers' reports of externalizing problems on the CBCL was $r(49) = .42$ ($p = .002$) from 30 to 36 months and $r(39) = .48$ ($p = .001$) from 36 to 42 months. Cross-time continuity of parents' reports on the ECBI was $r(142) = .58$ ($p < .001$) from 30 to 36 months and $r(111) = .64$ ($p < .001$) from 36 to 42 months. Cross-time continuity increased over time for parent-reported CBCL externalizing problems ($t = -3.47$, $p < .001$), but not for secondary caregiver-reported CBCL externalizing problems ($t = -0.49$, $p = .629$) or parent-reported ECBI behavior problems ($t = -1.18$, $p = .240$).

Despite relatively high cross-time continuity in externalizing problems, cross-informant correlations were much weaker (.03 to .31). Correlations across raters of CBCL externalizing problems were $r(96) = .17$ ($p = .089$), $r(78) = .03$ ($p = .777$), and $r(74) = .31$ ($p = .006$) at 30, 36, and 42 months, respectively. The internal consistency of externalizing ratings on the CBCL, as measured by Cronbach's alpha, was $\alpha = .89$, .89, and .89 for parents' ratings at 30, 36, and 42 months, respectively, and $\alpha = .93$, .89, and .92 for secondary caregivers' ratings. The internal consistency of parents' ratings of behavior problems on the ECBI was $\alpha = .88$, .86, and .88 at 30, 36, and 42 months, respectively. Descriptive statistics (means, standard deviations, minima, and maxima) of the self-regulation tasks are presented in Table 2.7. The correlations among the ratings of externalizing problems are in Table 2.8.

Table 2.7: All Studies: Descriptive Statistics of the Ratings of Children's Externalizing Problems.

	CBCL			CBCL Secondary			ECBI		
Age (mo)	30	36	42	30	36	42	30	36	42
N	273.00	183.00	165.00	102.00	82.00	77.00	207.00	147.00	130.00
M	12.12	12.42	11.88	8.07	7.94	7.29	107.10	109.13	110.07
SD	6.81	6.81	6.89	7.25	6.56	7.27	22.67	21.46	25.70
min	0.00	0.00	0.00	0.00	0.00	0.00	41.00	44.00	33.00
max	32.00	30.00	39.00	28.00	27.00	33.00	174.00	161.00	172.00

Note. "mo" = months. "All Studies" refers to the full sample of 336 families who were part of the larger study (i.e., not just those who were recruited for the EEG procedures).

2.1.2.4 Missingness

We examined whether children's data missingness differed systematically as a function of other variables, including temperament (as reported by parents on the Child Behavior Questionnaire, CBQ; Rothbart, Ahadi, Hershey, & Fisher, 2001), externalizing problems, age, sex, and SES. Compared to children who provided usable electrophysiological data, children who did not provide usable data did not differ in terms of parent-reported externalizing problems ($t[25.54] = -0.87$, $p = .390$), fearful temperament ($t[29.35] = 0.61$, $p = .545$), impulsive temperament ($t[13.51] = 0.48$, $p = .637$), sex ($\chi^2[1] = 0.45$, $p = .740$), or SES ($t[15.22] = -0.94$, $p = .364$). Moreover, several children ($n = 4$) who provided usable data had scores above the normed 80th percentile on externalizing problems (i.e., T-score > 58). On the other hand, younger children tended to be more likely to be missing electrophysiological data than older children ($t[26.09] = -1.92$, $p = .066$). This is not surprising because younger children may be less likely to sit still during the tasks. As a result, we included the child's age as a covariate in the clustered regression models. With the exception of missingness by the child's age, however, there was no evidence of any systematic missingness in

TABLE 2.8: All Studies: Pearson Correlations of the Ratings of Children's Externalizing Problems.

	CBCL			CBCL Secondary			ECBI		
Age (mo)	30	36	42	30	36	42	30	36	42
CBCL 30	–								
CBCL 36	.59***	–							
CBCL 42	.56***	.75***	–						
CBCL Secondary 30	.17†	−.12	−.05	–					
CBCL Secondary 36	.03	.03	.09	.42**	–				
CBCL Secondary 42	.15	.23†	.31**	.50**	.48**	–			
ECBI 30	.63***	.44***	.49***	.20†	.18	.17	–		
ECBI 36	.42***	.52***	.41***	−.02	.10	.11	.58***	–	
ECBI 42	.47***	.53***	.58***	.16	.13	−.05	.59***	.64***	–

Note. $^{\dagger}p < .10$. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$. "mo" = months. "All Studies" refers to the full sample of 336 families who were part of the larger study (i.e., not just those who were recruited for the EEG procedures). Correlations are two-tailed. *N*s range from 41 to 207.

variables of interest that would compromise the representativeness of the sample and generalizability of findings. There were not enough children with secondary caregiver reports and missing electrophysiological data to test whether there was systematic missingness as a function of secondary caregiver-reported scales.

2.1.3 Procedure

This study was part of a larger ongoing longitudinal study that follows children and their families and collects measures on demographics, parenting, sleep, temperament, cognitive development, self-regulation, and adjustment in the context of two home visits and two lab visits at multiple ages of assessment. The full procedure was as follows: (1) the questionnaires were distributed in the initial home visit (1 hour). (2) Within the next several days, there was a second home visit (2 hours) to collect information on the child's sleep routine and other measures pertaining to the main study. (3) Approximately one week following the initial home visit, a battery of self-regulation behavioral tasks was conducted in the first lab visit (2 hours). (4) One week following the first lab visit, families had a second lab visit to collect EEG measures (1 hour). The sample was recruited from a community sample representing a full range of risk (important for investigations of individual differences), in order to provide adequate statistical power for the proposed models.

In total, the behavioral battery included 19 tasks related to parent–child interaction, inhibitory control, sustained attention, motor inhibition, regulation in reward situations, and emotion regulation. The present study focused only on the self-regulation tasks involving inhibitory control (Bird/Alligator, Grass/Snow, and Shape Stroop) and sustained attention (Token Sort and Sustained Play Attention). We focused on the inhibitory control and

sustained attention tasks (a) to limit the scope of the possible questions and (b) because, as has been described, we expect performance on these tasks to be particularly related to externalizing problems and to neural functioning during go/no-go and oddball tasks.

The procedure during the EEG lab visit was as follows: First, the child played a puzzle with the research assistant to establish rapport and help the child feel comfortable. Second, the child chose a video to watch among a set of developmentally-appropriate videos. Third, the child watched the video while the EEG net was applied and electrode impedances were adjusted. Fourth, the child played the two EEG tasks. Finally, 5 minutes of baseline EEG were recorded while children continued watching the video they chose.

2.1.4 Inclusion of Incorrect Trials in Electrophysiological Analyses

In order to retain most children in the electrophysiological analyses, we included their incorrect trials in the subject averages to be used in the analyses. Although including incorrect trials in subject averages is not traditional in the neurophysiological literature, we did so for several key theoretical reasons. From a cognitive perspective, just because a child does not respond correctly in a given trial does not mean they were not trying—in fact we think it is likely that their brains were still processing the information. From a developmental perspective, excluding incorrect trials would systematically exclude the youngest children. It is important to establish a baseline to see development of behavior and neural activity over time, and excluding the youngest children would preclude establishing a meaningful baseline of behavior and neural activity. From a clinical science perspective, excluding incorrect trials would likely exclude those children with the most behavior problems (exactly who we are most interested in). From a statistical perspective, because the predictor

(neural activity) and outcomes (self-regulation and behavior problems) are from completely different measurement sources, they do not share source or method variance. As a result, including incorrect trials would not increase the shared variance and would not artifactually increase the likelihood of detecting an association. The bottom line is that it is necessary to accommodate the population of interest and including incorrect trials would, if anything, add noise to waveforms, which would make it *less* likely to detect associations with other variables.

Including incorrect trials in the subject averages for analysis was necessary to accommodate the population of interest. This approach, though novel, is consistent with a precedent in oddball paradigms in which participants respond to rare stimuli by keeping a silent count of the rare stimuli encountered throughout the trial block. Many of these studies include all trials in a block based on whether or not the overall number of correct responses in that block surpassed an overall correct response threshold, thereby including individual trials in the grand average in which a behavioral response was not made (Habeych, Charles, Sclabassi, Kirisci, & Tarter, 2005; Martin, Barajas, Fernandez, & Torres, 1988). Martin, Barajas, Fernandez, and Torres (1988) attributed these "errors" to children's confusion in counting (difficulties with the demands of the task) rather than perceptual errors. In the present study, every toddler, except for one, made correct behavioral responses to the target stimuli, but not on every trial. This is consistent with our understanding of sustained attention as a developing skill in toddlerhood. This likely reflects variability in toddlers' capacity to respond consistently on command in the context of a task requiring focal attention (difficulties with the demands of the task), rather than a deficit in the processing of

the deviant stimuli. To compare our approach to a more traditional approach, we also conducted our analyses excluding children who did not have a sufficient number of artifact-free target trials with a behavioral response (Hoyniak, Petersen, McQuillan, Staples, & Bates, in press). Excluding children based on their ability to consistently sustain attention and make a behavioral response introduced unacceptable systematic missingness to our data. The youngest children had the fewest artifact-free target trials with a behavioral response such that children under age three were systematically excluded. Because toddlerhood is a sensitive period of development for the capacity to sustain attention and inhibit prepotent responses, this excluded a highly interesting subset of children and reduced our ability to describe development in toddlerhood.

Researchers who study cognitive development in early childhood distinguish between ability and performance, observing that variability in performance accuracy does not necessarily indicate variability in the underlying cognitive process (L. B. Smith & Katz, 1996). Rather, variable performance can reflect a stable ability (Medin & Ortony, 1989). For example, we have evidence that children in the sample properly categorized the stimuli even though they did not consistently make a correct behavioral response. Children responded more frequently to the relevant target/go stimulus than the nontarget/no-go stimulus in both tasks. Thus, the children in our sample who showed variability in their behavioral response still recognized and processed the stimuli even in the absence of a consistent behavioral response. In sum, we adapted our approach for the population and research questions of interest to increase generalizability and feasibility in ways that are consistent with theory.

2.1.5 Statistical Analysis

2.1.5.1 Statistical Models

To test the hypothesis that ERP components predict the development of externalizing problems, we used Pearson correlation and multiple regression. Eight children had measurements at 2 occasions. An assumption of Pearson correlation and multiple regression is that observations are independent (i.e., independent residuals; Osborne & Waters, 2002). Because of the longitudinal design, there was dependency in observations owing to multiple assessments for the same participant. To test the hypotheses, we first examined Pearson correlations with the data in long form where each measurement occasion is treated as a different participant (only for description of the general association between variables). For those correlations that were consistent with the hypotheses, the associations were (1) examined with Spearman's rank correlation (rho) that is a nonparametric estimate of correlation that is robust to outliers, and (2) probed in multiple regression models that used a cluster variable to account for the dependency in the data owing to multiple measurement occasions nested within the same participant (i.e., clustered regression). Clustered regression models were fit in the rms package (Harrell Jr., 2014) in R version 3.0.2 (R Core Team, 2013), which calculates robust standard errors using a robust (Huber-White sandwich) estimator of the covariance matrix (Huber, 1967; White, 1980).[2] Sandwich estimators are widely used to account for data dependency in regression models (for an example using sandwich

[2]It is known as a "sandwich" estimator because the formula metaphorically includes a piece of meat (the inverse variance of the parameter estimates) between two pieces of bread (the model-based variance estimate).

estimators in the context of longitudinal neuroimaging, see Guillaume, Hua, Thompson, Waldorp, & Nichols, 2014).

When we had adequate observations to include covariates in the regression models, we included covariates for sex, age, the number of bad channels, the number of trials kept in the condition of interest (no-go or target), and behavioral percent correct in the condition of interest. Nonlinear (quadratic) associations between N2/P3b amplitudes and adjustment were examined, as well. We fit quadratic regression models by adding a quadratic term for ERP amplitudes to the linear regression model. Linear and quadratic model fits were compared using nested model deviance tests based on log-likelihood. Quadratic regression models that fit significantly better than linear regression models were probed with scatterplots. Estimates in parentheses for all r-, t-, χ^2-tests reflect degrees of freedom. For independent samples t-tests, we used Welch's t-test, which does not assume equal variances across groups (i.e., the variance is estimated separately for both groups and the Welch–Satterthwaite modification to the degrees of freedom is used; Satterthwaite, 1946; Welch, 1947).

To test the mediational hypotheses, we examined mediation models in structural equation modeling (SEM) using Mplus version 6.12 (L. K. Muthén & B. O. Muthén, 2011). Missing data in SEM can be handled by using full information maximum likelihood (FIML) estimates, which provide unbiased estimates when the data are missing at random, even if not completely at random (Schafer & Graham, 2002). Indirect effects were tested by bootstrapping bias-corrected 95% confidence intervals from 1,000 bootstrap samples, as recommended by Shrout and Bolger (2002) for tests of mediation with small sample sizes.

Because the present study examined a fairly novel population in the context of investigating neurophysiological correlates of self-regulation and externalizing behavior, we followed a somewhat exploratory approach (yet we tested specific hypotheses). Therefore, we did not control for multiple testing. Nevertheless, all effects were examined with two-tailed tests with an alpha level of .05 (for exploratory purposes, however, we also note trend-level effects of $p < .10$). To limit the family-wise Type I error rate, we further examined (i.e., with clustered regression and mediational tests) only those associations (a) that were consistent with hypotheses or (b) that showed patterns of associations across measures.

2.1.5.2 Power

ERPs tend to have less measurement error and higher reliability than behavioral measures (Räikkönen et al., 2003), resulting in larger effect sizes that permit smaller sample sizes. Sixteen prior studies examining the association between the N2 ERP and self-regulation or externalizing problems had a medium effect size ($d = 0.46$) on average (see Table 1.1). With usable data on 27 children, we would have low power (.21) to detect a simple bivariate association of this magnitude at a given age ($\alpha = .05$, two tailed). However, with longitudinal data, we would have somewhat higher power (.45) to detect an association of this magnitude (Scherbaum & Ferreter, 2009). Sample sizes in analyses examining correlations between children's neurophysiology and their sustained attention scores were fairly small because only a later part of the sample (17 children, 63%) was given the opportunity to complete the sustained attention tasks. Also, fewer secondary caregivers than parents reported on children's behavior problems because some families did not have a secondary caregiver that spent 10 or more hours in the preceding month with the child. As a result,

power is likely low to detect associations between neurophysiology and sustained attention and secondary caregiver-reported behavior problems in Study 1. Because correlations are particularly sensitive to extreme values, especially in cases of small sample sizes, we present the correlational inferences with caution.

2.1.6 Preliminary Findings

Our preliminary findings support the hypotheses that longer N2 and P3b latencies predict self-regulation deficits and externalizing problems. Children with longer no-go N2 latencies showed poorer self-regulation in the Bird/Alligator inhibitory control task ($r = -.62$, $p = .02$; Petersen et al., 2013c) and more parent-rated externalizing problems ($r = .50$, $p = .04$; Petersen et al., 2013a). Children with longer oddball P3b latencies showed more parent-rated attention problems ($r = .59$, $p = .07$; Petersen et al., 2013b), and smaller P3b amplitudes were associated with more externalizing problems ($r = -.50$, $p = .05$; Petersen et al., 2013b). In addition, children with less frontal alpha power in the Bird/Alligator ($r = -.43$, $p = .012$) and oddball ($r = -.67$, $p = .001$) tasks showed more parent-rated externalizing problems (Petersen, Hoyniak, Bates, Staples, & Molfese, 2015). Moreover, our findings suggest a candidate mediating neural process by which stressors such as sleep deficits may lead to behavior problems. Children with irregular night-to-night sleep (in terms of duration and timing) showed longer P3b latencies, and children with longer P3b latencies showed poorer sustained attention (Hoyniak et al., in press).

2.2 Results

2.2.1 Association Between Neurophysiology and Self-Regulation

2.2.1.0.1 ERPs and Self-Regulation. Pearson correlations of children's ERP components with their self-regulation are in Table 2.9. Few associations between ERPs and self-regulation scores were observed. Larger target P3b amplitudes ($r[16] = .41$, $p = .095$, see Figure 2.26) and larger P3b amplitude difference scores (i.e., having a larger target P3b amplitude than frequent P3b amplitude; $r[16] = .40$, $p = .098$, see Figure 2.27) were marginally significantly associated with better performance on the Bird/Alligator inhibitory control task (i.e., the behavioral task). Target P3b amplitudes ($p = .213$) and P3 amplitude difference scores ($p = .162$) were no longer significantly associated with performance on the Bird/Alligator task when examining Spearman's rho, suggesting that the association may have been accounted for, in part, by outliers. Clustered regression models examining the association of target P3b amplitudes and P3 amplitude difference scores with performance on the Bird/Alligator task are in Table 2.10. The target P3b amplitude and P3 amplitude difference score did not remain associated with Bird/Alligator inhibitory control after accounting for the nesting of longitudinal data and controlling for covariates. The only significant predictor of performance on the Bird/Alligator task in these models was the behavioral accuracy on the target trials (better accuracy on the target trials was associated with better performance on the Bird/Alligator task).

Larger (i.e., more negative) no-go than go amplitudes were associated with better performance on Shape Stroop ($r[30] = -.39$, $p = .028$, see Figure 2.28). N2 amplitude difference scores remained associated with performance on Shape Stroop when examining Spearman's

TABLE 2.9: Study 1: Pearson Correlations of Children's ERP Components with their Self-Regulation.

	P3b					N2				
	Tgt Amp	Frq Amp	Amp Diff	Tgt Lat	Frq Lat	Go Amp	No-Go Amp	Amp Diff	Go Lat	No-Go Lat
Bird/Alligator	.41†	−.26	.40†	.02	−.26	.01	−.07	−.07	.18	−.24
Shape Stroop	.07	.16	−.03	−.25	−.08	.31†	−.14	−.39*	.26	−.25
Grass/Snow	.29	−.14	.27	.43†	−.04	.05	.02	−.03	−.16	−.06
Token Sort	.03	−.20	.11	.05	−.19	−.11	−.33	−.18	−.10	−.12
Sustained Play Attention	.42	.08	.26	−.29	.11	.15	.12	−.03	−.10	−.09
Bird/Alligator (ERP)	−.09	.06	−.09	.09	−.30	.04	−.27	−.24	−.05	.13
Oddball	.34	−.12	.29	−.07	−.24	.28	.17	−.12	.22	−.15

Note. "Amp" = amplitude, "Lat" = latencies, "Diff" = difference, "Tgt" = target, "Frq" = frequent. Amplitudes are in microvolts, latencies are in milliseconds. P3b amplitude difference reflects target P3b amplitude − frequent P3b amplitude. N2 amplitude difference reflects no-go N2 amplitude − go N2 amplitude. Correlations are two-tailed.

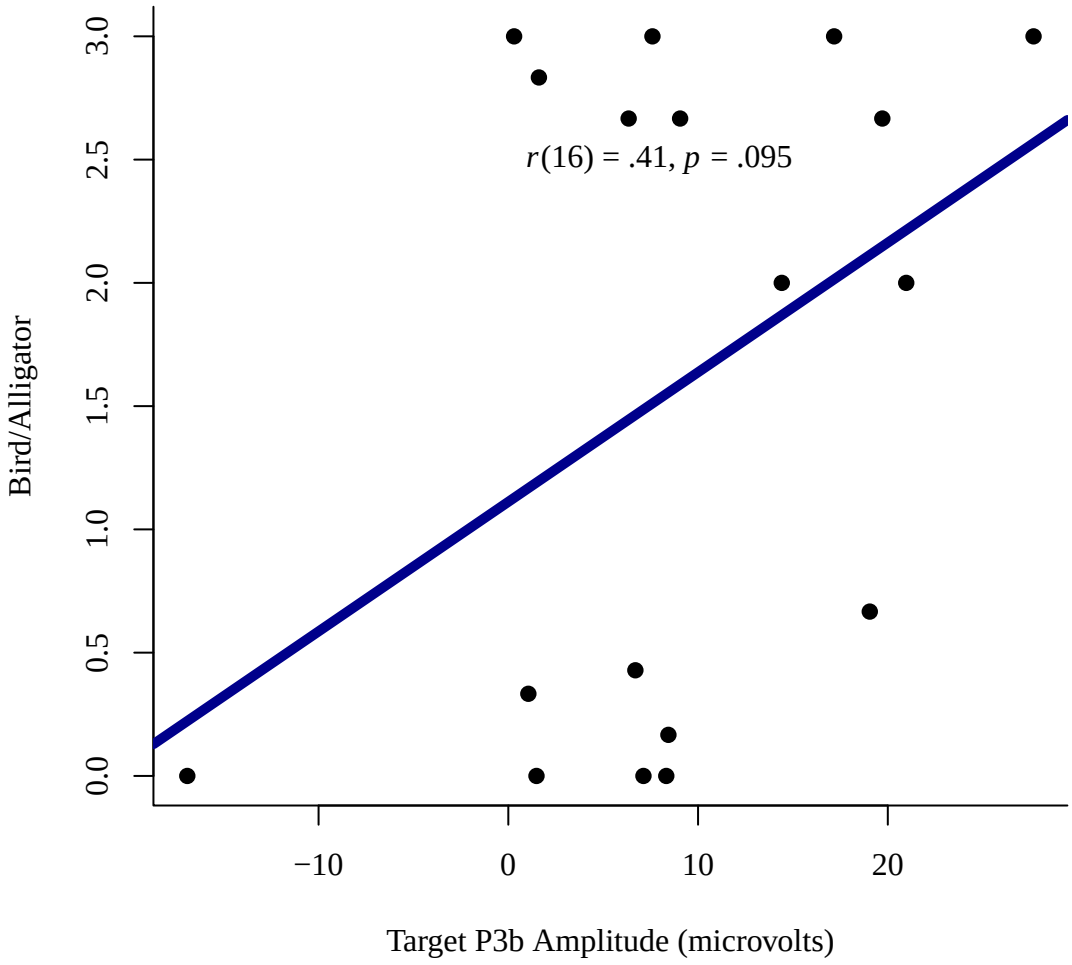

FIGURE 2.26: Association between target P3b amplitude and performance on the Bird/Alligator inhibitory control task.

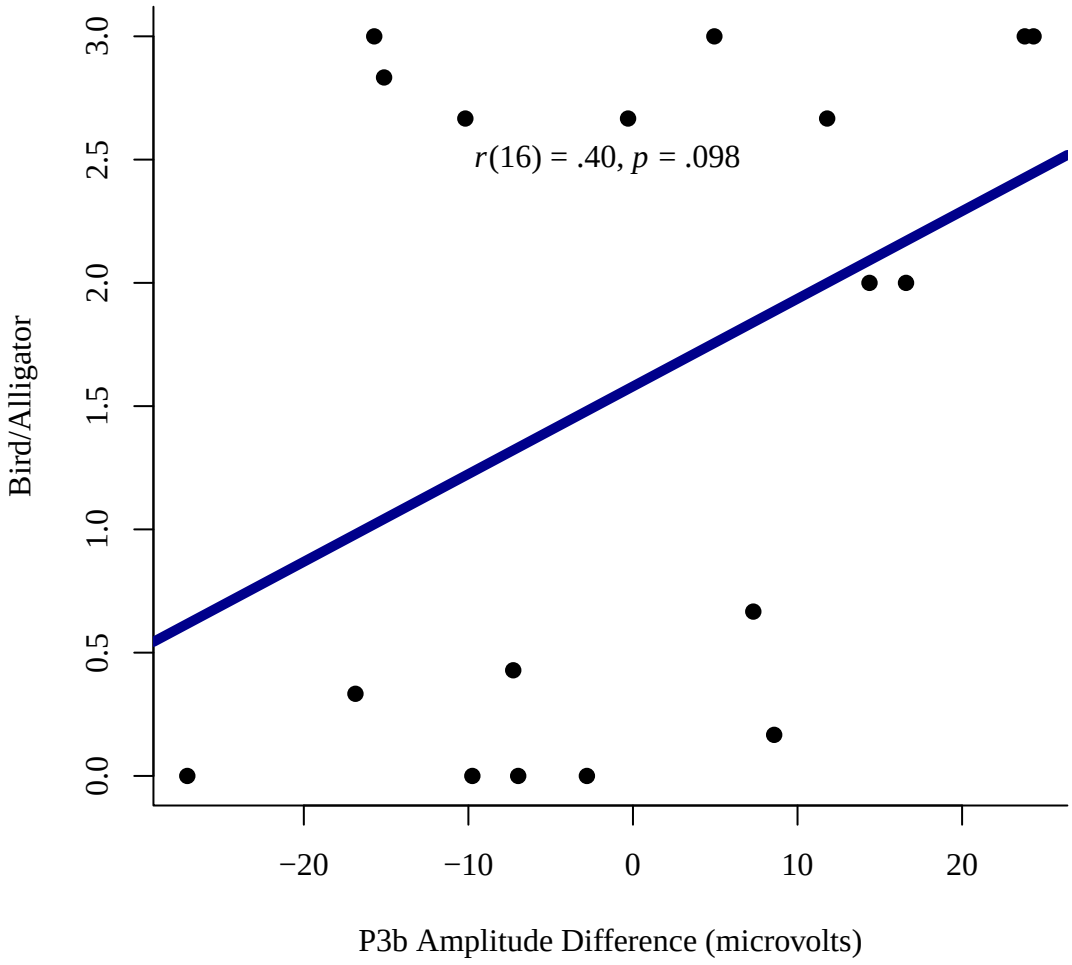

FIGURE 2.27: Association between P3b amplitude difference (target P3 amplitude − frequent P3 amplitude) and performance on the Bird/Alligator inhibitory control task.

TABLE 2.10: Study 1: Clustered Regression Examining Association Between P3b Amplitude and Performance on Bird/Alligator (Inhibitory Control).

	Dependent variable:	
	Bird/Alligator (Inhibitory Control)	
Intercept	11.125^{***} (3.225)	10.318^{**} (3.177)
Target P3b Amplitude	−0.011 (0.015)	
Target P3b Amplitude Difference		0.003 (0.009)
Sex	−0.550 (0.557)	−0.435 (0.533)
Age	-2.539^{***} (0.745)	-2.312^{**} (0.771)
Number of Bad Channels	-0.351^{***} (0.095)	-0.323^{***} (0.089)
Number of Target Trials Kept	$-0.120^{\dagger}$ (0.076)	$-0.127^{\dagger}$ (0.074)
Behavioral Percent Correct on Target Trials	0.061^{***} (0.010)	0.057^{***} (0.009)
Observations	18	18
R^2	0.730	0.727
Adjusted R^2	0.582	0.578

Note. Age in years. Sex is coded as male = 0, female = 1. $^{\dagger}p < .10$. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

rho ($p = .035$). A clustered regression model examining the association between N2 amplitude difference scores and performance on Shape Stroop is in Table 2.11. The N2 amplitude difference score remained associated with Shape Stroop self-regulation after accounting for the nesting of longitudinal data and controlling for covariates.

Inconsistent with hypotheses, longer target P3b latencies were marginally significantly associated with *better* performance on the Grass/Snow task. No associations between N2/P3b amplitudes and self-regulation scores showed better quadratic than linear fit.

2.2.1.0.2 EEG and Self-Regulation. Pearson correlations of children's EEG power values and asymmetry scores with their self-regulation are in Table 2.12. Greater frontal alpha power in the Bird/Alligator ERP task was marginally significantly associated with better no-go performance on the same task ($r[32] = .33$, $p = .055$, see Figure 2.29). The association between frontal alpha power and performance on the Bird/Alligator ERP task did not remain statistically significant when examining Spearman's rho ($p = .548$), suggesting that the association may have owed, in part, to outliers. Moreover, frontal alpha power was

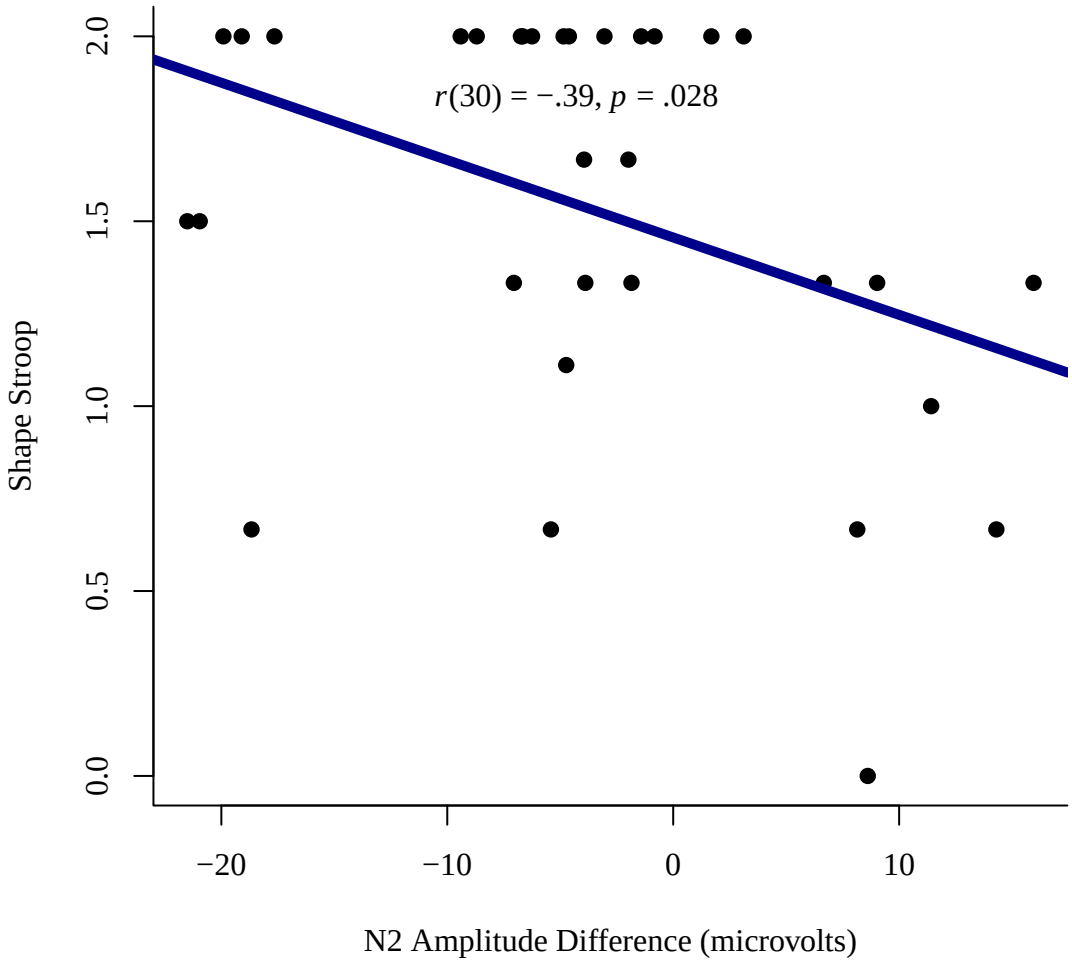

FIGURE 2.28: Association between N2 amplitude difference (no-go N2 amplitude − go N2 amplitude) and performance on the Shape Stroop inhibitory control task.

TABLE 2.11: Study 1: Clustered Regression Examining Association Between N2 Amplitude Difference and Performance on Shape Stroop.

	Dependent variable:
	Shape Stroop
Intercept	−0.445 (1.130)
N2 Amplitude Difference	−0.017* (0.008)
Sex	−0.007 (0.214)
Age	0.690* (0.323)
Number of Bad Channels	−0.010 (0.032)
Number of No-Go Trials Kept	0.005 (0.017)
Behavioral Percent Correct on No-Go Trials	−0.0003 (0.004)
Observations	32
R^2	0.280
Adjusted R^2	0.107

Note. $^{\dagger}p < .10$. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

not statistically significantly associated with performance on the Bird/Alligator ERP task after accounting for the nesting of longitudinal data and controlling for covariates (see Table 2.13). The only significant predictor of no-go performance was the child's age—children performed better on the Bird/Alligator ERP task with age.

Left frontal asymmetry (greater left than right frontal activation in the alpha frequency band) was marginally significantly associated with worse performance on Shape Stroop ($r[18] = -.42$, $p = .067$, see Figure 2.30). The association between left frontal asymmetry and poorer performance on Shape Stroop remained marginally significant when examining Spearman's rho ($p = .079$). Left frontal asymmetry remained associated with poorer performance on Shape Stroop after accounting for the nesting of longitudinal data and controlling for covariates (see Table 2.14). Inconsistent with hypotheses, however, left frontal asymmetry was significantly associated with *better* performance on the Sustained Play Attention task.

2.2.1.0.3 Time-Frequency Neurophysiology and Self-Regulation. Pearson correlations of children's time-frequency values with their self-regulation are in Table 2.12. Inconsistent with hypotheses, greater N2-related frontal theta activity in the Bird/Alligator ERP task was associated with worse performance on the target trials of the oddball task ($r[28] = -.44$, $p = .014$, see Figure 2.31), and was marginally significantly associated with worse performance on the Bird/Alligator inhibitory control task ($r[27] = -.36$, $p = .057$, see Figure 2.32). The association of N2-related frontal theta activity with performance on the oddball task ($p = .013$) and Bird/Alligator inhibitory control task ($p = .024$)

TABLE 2.12: Study 1: Pearson Correlations of Children's EEG and Time-Frequency Components with their Self-Regulation.

	Oddball			Bird/Alligator		
	Frontal Power	Frontal Asymmetry	Frontal TF	Frontal Power	Frontal Asymmetry	Frontal TF
Bird/Alligator	.25	−.26	−.14	−.08	.12	−.36†
Shape Stroop	.35	−.42†	−.01	−.02	−.09	−.18
Grass/Snow	−.19	−.18	.05	−.17	.19	−.12
Token Sort	.12	.08	.26	.22	.01	−.30
Sustained Play Attention	.02	.56*	.26	.02	.11	−.30
Bird/Alligator (ERP)	.12	−.16	−.27	.33†	−.17	−.17
Oddball	.30	−.16	.15	−.07	−.25	−.44*

Note. "TF" = time-frequency activity corresponding to timing of P3b (oddball) or N2 (Bird/Alligator), with values in decibels. Power values were log-transformed. Frontal power and asymmetry in alpha frequency range. Frontal time-frequency in theta frequency range. Frontal asymmetry reflects right frontal alpha power − left frontal alpha power (i.e., higher values reflect left frontal asymmetry in alpha frequency range). Correlations are two-tailed.

Table 2.13: Study 1: Clustered Regression Examining Association Between Frontal Alpha Power (Bird/Alligator) and Performance on Bird/Alligator (ERP).

	Dependent variable:
	Bird/Alligator ERP No-Go Percent Correct
Intercept	−41.385 (61.836)
Frontal Alpha Power (Bird/Alligator)	15.320 (10.992)
Sex	7.258 (7.318)
Age	33.117** (11.973)
Number of Bad Channels	−1.690 (1.223)
Number of No-Go Trials Kept	−0.666 (0.817)
Observations	34
R^2	0.332
Adjusted R^2	0.213

Note. Age in years. Sex is coded as male = 0, female = 1. $^{\dagger}p < .10$. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

Table 2.14: Study 1: Clustered Regression Examining Association Between Left Frontal Asymmetry (Oddball) and Performance on Shape Stroop.

	Dependent variable:
	Shape Stroop
Intercept	3.439† (2.013)
Left Frontal Asymmetry (Oddball)	−0.400 (0.343)
Sex	−0.179 (0.221)
Age	−0.358 (0.510)
Number of Bad Channels	−0.065 (0.051)
Number of Target Trials Kept	−0.071 (0.050)
Behavioral Percent Correct on Target Trials	0.016* (0.008)
Observations	19
R^2	0.513
Adjusted R^2	0.269

Note. Age in years. Sex is coded as male = 0, female = 1. $^{\dagger}p < .10$. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

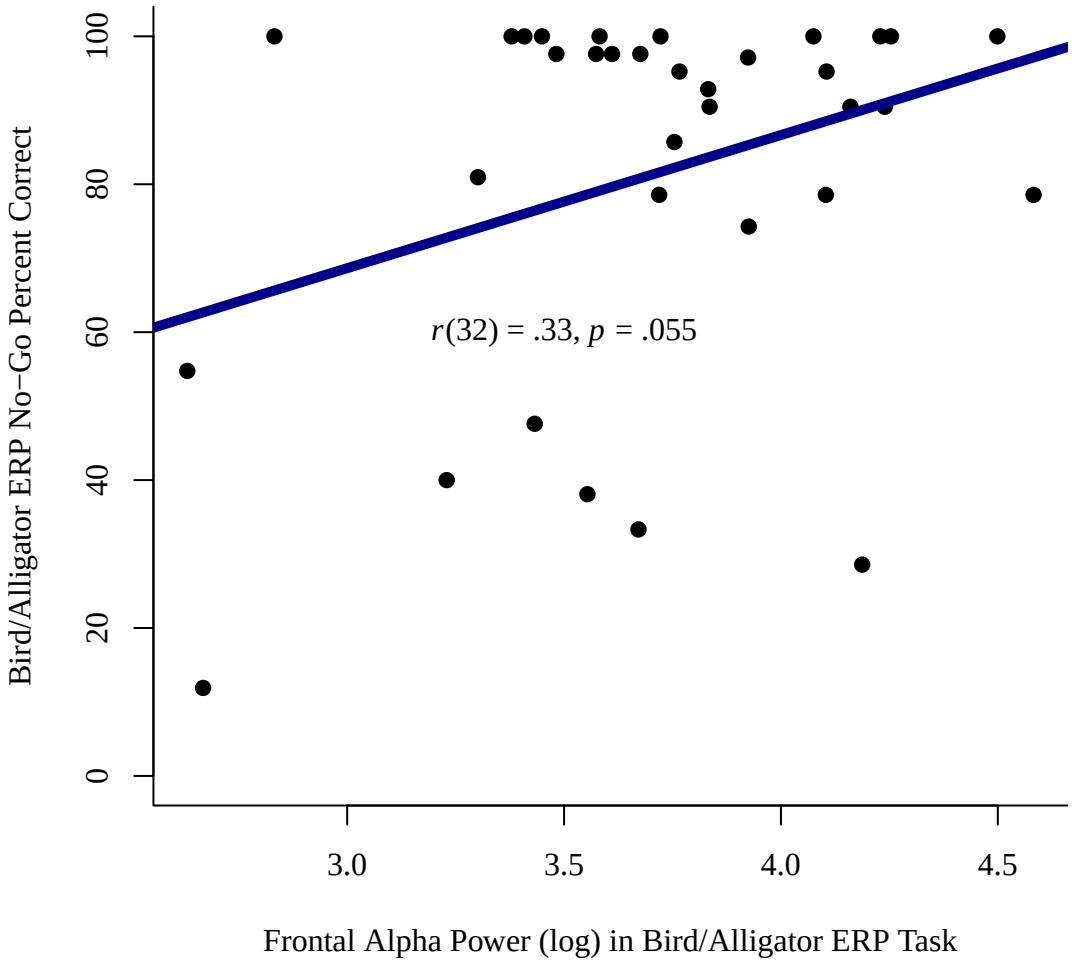

FIGURE 2.29: Association between frontal alpha power in the Bird/Alligator task and performance on no-go trials of the Bird/Alligator ERP task.

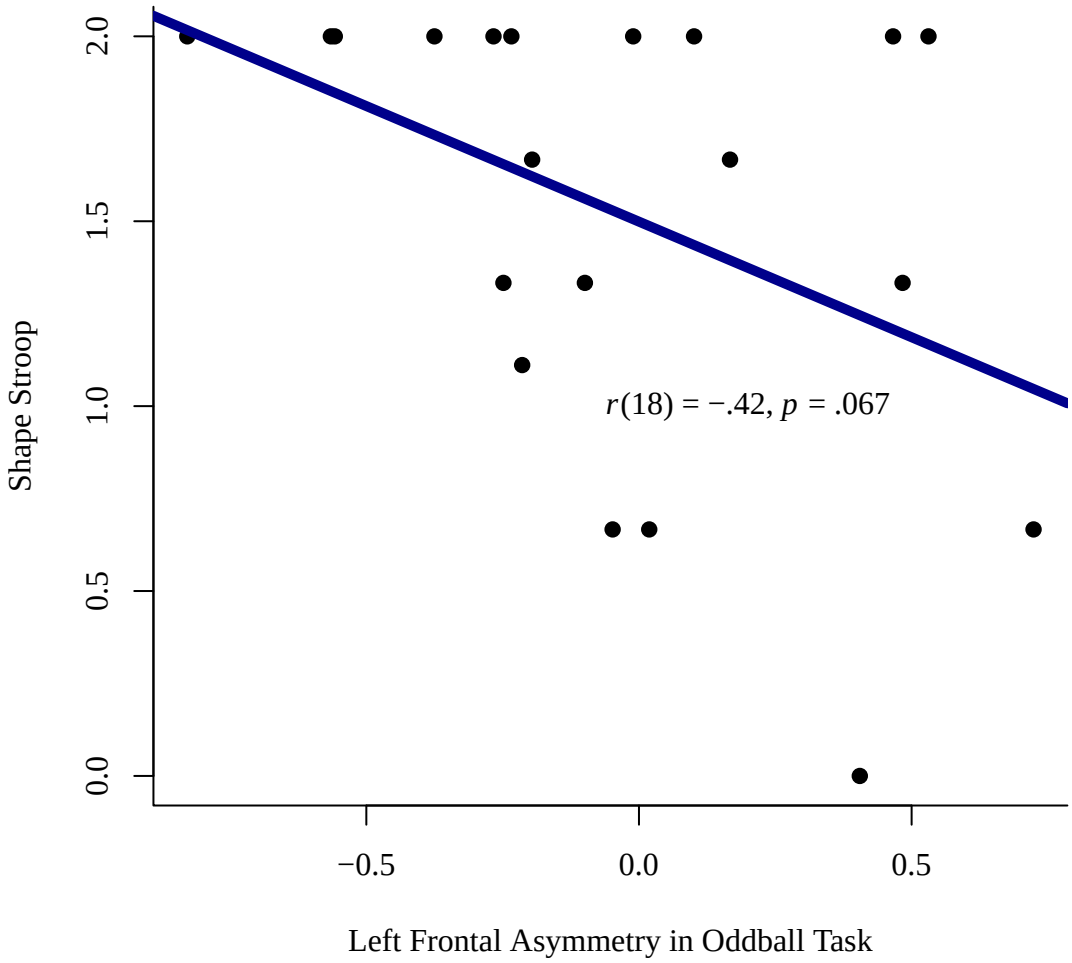

FIGURE 2.30: Association between left frontal asymmetry in the oddball task and performance on the Shape Stroop inhibitory control task.

remained significant when examining Spearman's rho. N2-related frontal theta activity remained associated with performance on the oddball and Bird/Alligator inhibitory control tasks after accounting for the nesting of longitudinal data and controlling for covariates (see Table 2.15).

TABLE 2.15: Study 1: Clustered Regression Examining Association of N2-Related Frontal Theta Activity with Performance on the Oddball and Bird/Alligator (Inhibitory Control) Tasks.

	Dependent variable:	
	Oddball Target Percent Correct	Bird/Alligator (Inhibitory Control)
Intercept	-65.485^{*} (31.491)	-2.080 (2.808)
N2-Related Frontal Theta Activity	-1.735^{**} (0.618)	-0.104^{*} (0.048)
Sex	-9.509 (8.372)	-0.605 (0.379)
Age	46.933^{***} (11.101)	$1.308^{\dagger}$ (0.783)
Number of Bad Channels	$-1.646^{\dagger}$ (0.884)	$-0.098^{\dagger}$ (0.058)
Number of No-Go Trials Kept	0.065 (0.814)	0.028 (0.031)
Behavioral Percent Correct on No-Go Trials	-0.094 (0.124)	0.007 (0.008)
Observations	30	29
R^2	0.562	0.398
Adjusted R^2	0.448	0.233

Note. Age in years. Sex is coded as male = 0, female = 1. $^{\dagger}p < .10$. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

2.2.2 Association Between Neurophysiology and Externalizing Problems

2.2.2.0.1 ERPs and Externalizing Problems. Pearson correlations of children's ERP components with their externalizing problems are in Table 2.16. Few associations were observed between children's ERP components and their externalizing problems. Smaller target P3b amplitudes (and a smaller difference between target and frequent P3b amplitudes) were marginally significantly associated with more secondary caregiver-reported externalizing problems ($r[3] = -.82$, $p = .090$, see Figure 2.33) on the CBCL. The association between P3b amplitudes and secondary caregiver-reported externalizing problems remained marginally significant when examining Spearman's rho ($p = .083$). A clustered regression model examining the association of target P3b amplitudes with externalizing problems is

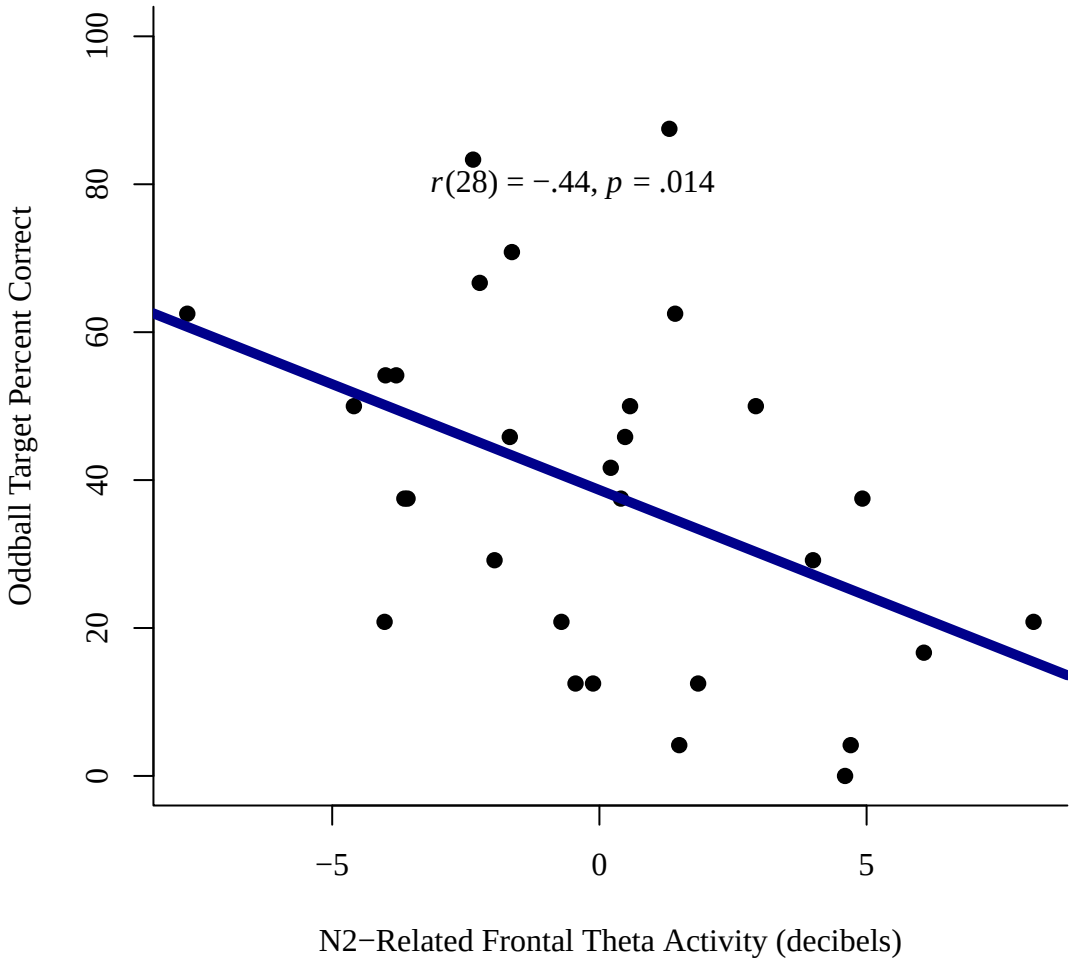

FIGURE 2.31: Association between N2-related frontal theta activity in the Bird/Alligator task and performance on the target trials of the oddball sustained attention task.

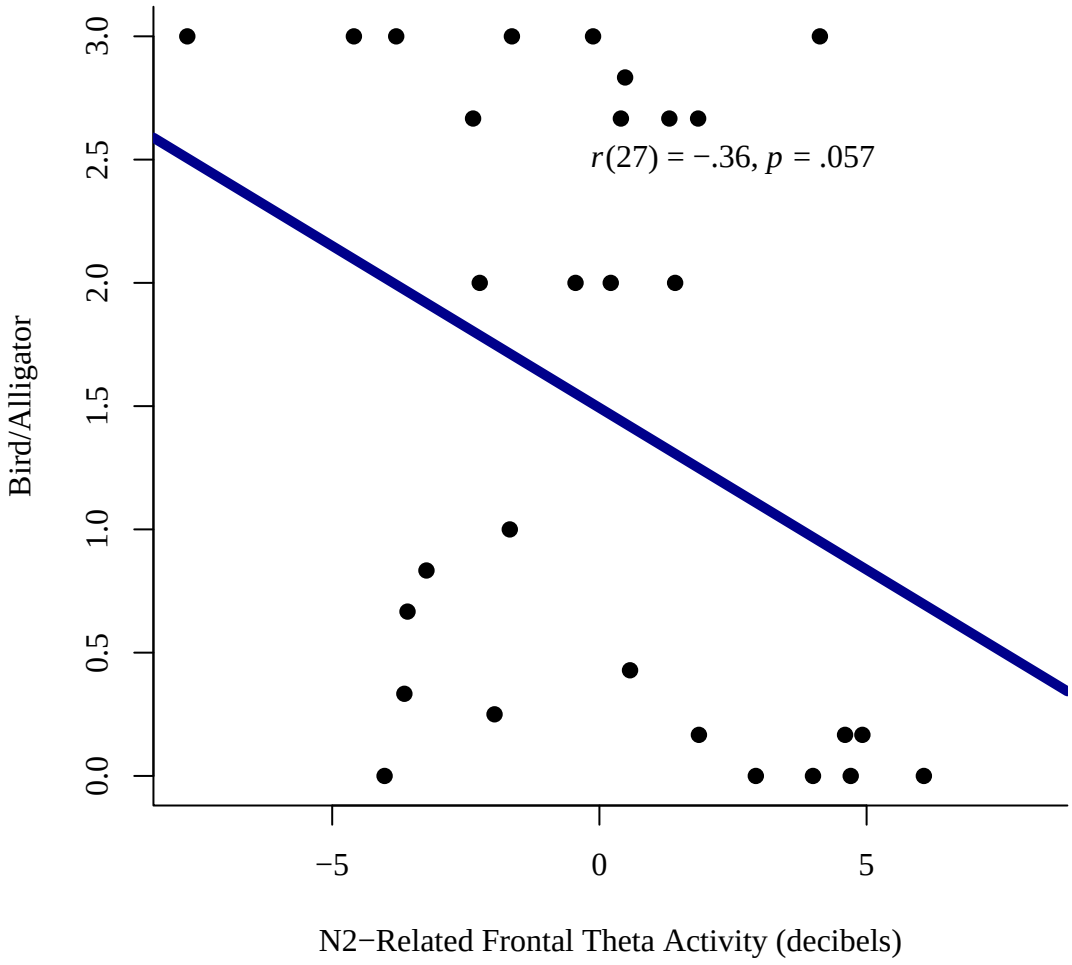

FIGURE 2.32: Association between N2-related frontal theta activity in the Bird/Alligator task and performance on the Bird/Alligator inhibitory control task.

in Table 2.17. The target P3b amplitude remained associated with externalizing problems even after accounting for the nesting of longitudinal data, although there were too few cases to control for covariates.

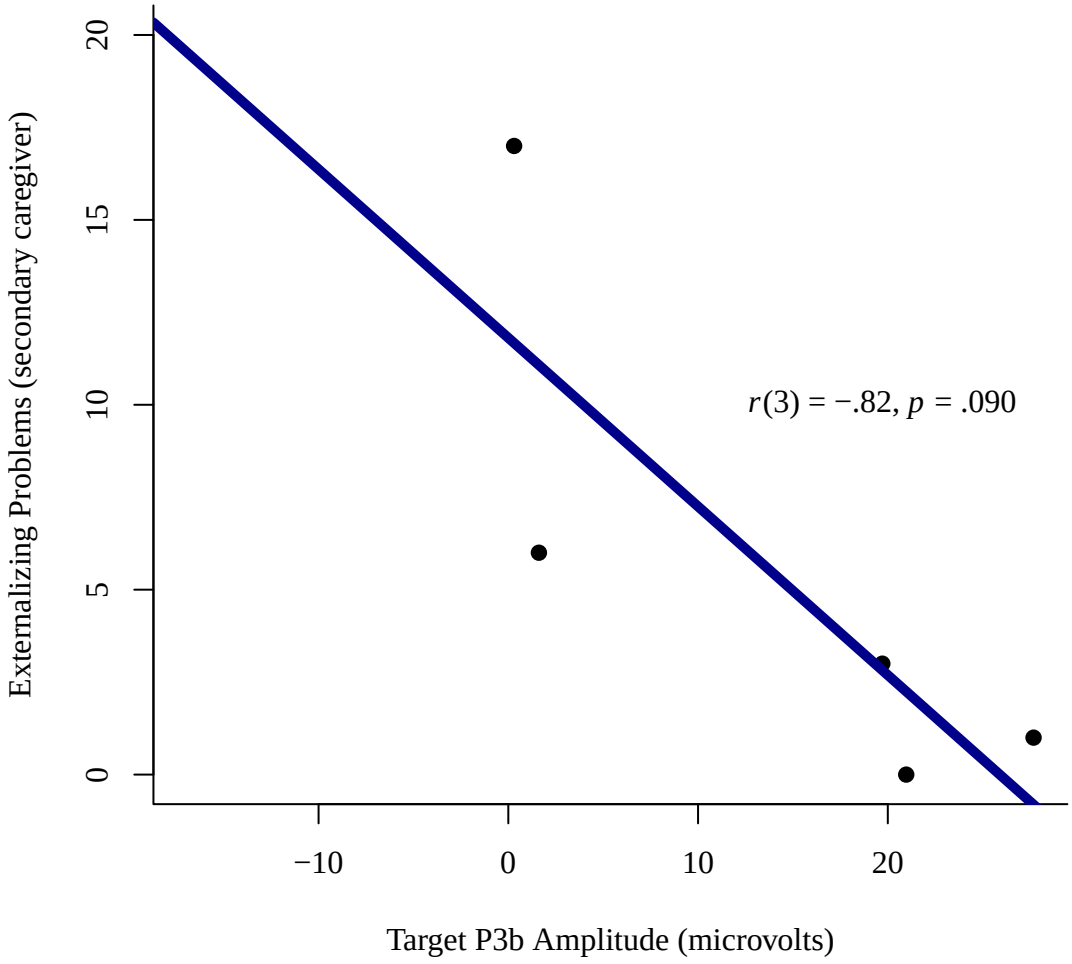

FIGURE 2.33: Association between target P3b amplitude and secondary caregiver-reported externalizing problems on the CBCL.

Longer target P3b latencies were marginally significantly associated with more parent-reported externalizing problems ($r[19] = .39$, $p = .085$, see Figure 2.34) and aggression

TABLE 2.16: Study 1: Pearson Correlations of Children's ERP Components with their Externalizing Problems.

	P3b					N2				
	Tgt Amp	Frq Amp	Amp Diff	Tgt Lat	Frq Lat	Go Amp	No-Go Amp	Amp Diff	Go Lat	No-Go Lat
CBCL EXT Parent	−.08	.17	−.14	.39†	−.04	−.07	.02	.08	.30†	.20
CBCL EXT Secondary	−.82†	.80	−.81†	.69	−.28	−.49	−.42	.12	.38	−.58
CBCL AGG Parent	−.12	.21	−.18	.38†	−.05	−.07	.02	.08	.31†	.24
CBCL AGG Secondary	−.79	.74	−.78	.65	−.28	−.38	−.40	.05	.38	−.49
CBCL ATT Parent	.11	−.02	.09	.32	.00	−.06	.00	.06	.15	−.01
CBCL ATT Secondary	−.33	.50	−.39	.40	−.05	−.67	−.17	.41	.06	−.61
ECBI Intensity Parent	−.29	−.06	−.18	.18	.14	−.48*	−.27	.20	.59**	.17

Note. "Amp" = amplitude, "Lat" = latencies, "Diff" = difference, "Tgt" = target, "Frq" = frequent, "EXT" = externalizing problems, "AGG" = aggression, "ATT" = attention problems. Amplitudes are in microvolts, latencies are in milliseconds. P3b amplitude difference reflects target P3b amplitude − frequent P3b amplitude. N2 amplitude difference reflects no-go N2 amplitude − go N2 amplitude. Correlations are two-tailed.

TABLE 2.17: Study 1: Clustered Regression Examining Association Between P3b Amplitude and Secondary Caregiver-Reported CBCL Externalizing Problems.

	Dependent variable:
	CBCL Externalizing Problems (Secondary)
Intercept	11.174^{***} (2.196)
Target P3b Amplitude	-0.520^{***} (0.127)
Observations	6
R^2	0.768
Adjusted R^2	0.711

Note. $^{\dagger}p < .10$. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

($r[19] = .38$, $p = .089$, see Figure 2.35). The associations of target P3b latencies with parent-reported externalizing problems ($p = .081$) and aggression ($p = .076$) remained marginally significant when examining Spearman's rho. Clustered regression models examining the association of target P3b latencies with parent-reported externalizing problems and with aggression are in Table 2.18. The target P3b latency remained associated with parent-reported externalizing problems and aggression even after accounting for the nesting of longitudinal data and after controlling for covariates.

TABLE 2.18: Study 1: Clustered Regression Examining Association of P3b Latency with Parent-Reported CBCL Externalizing Problems and Aggression.

	Dependent variable:	
	CBCL Externalizing Problems (Parent)	CBCL Aggression (Parent)
Intercept	-128.318^{**} (48.336)	-124.359^{**} (42.096)
Target P3b Latency	0.203^{***} (0.059)	0.178^{***} (0.051)
Sex	−3.040 (4.010)	−3.107 (3.273)
Age	1.029 (8.964)	4.575 (7.783)
Number of Bad Channels	0.247 (0.861)	0.328 (0.718)
Number of Target Trials Kept	0.230 (0.685)	0.332 (0.583)
Behavioral Percent Correct on Target Trials	0.048 (0.105)	0.003 (0.090)
Observations	20	20
R^2	0.283	0.323
Adjusted R^2	-0.047	0.010

Note. Age in years. Sex is coded as male = 0, female = 1. $^{\dagger}p < .10$. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

Neither amplitudes nor latencies of the no-go N2 were significantly associated with externalizing problems. Neither target P3b nor no-go N2 amplitudes showed better quadratic

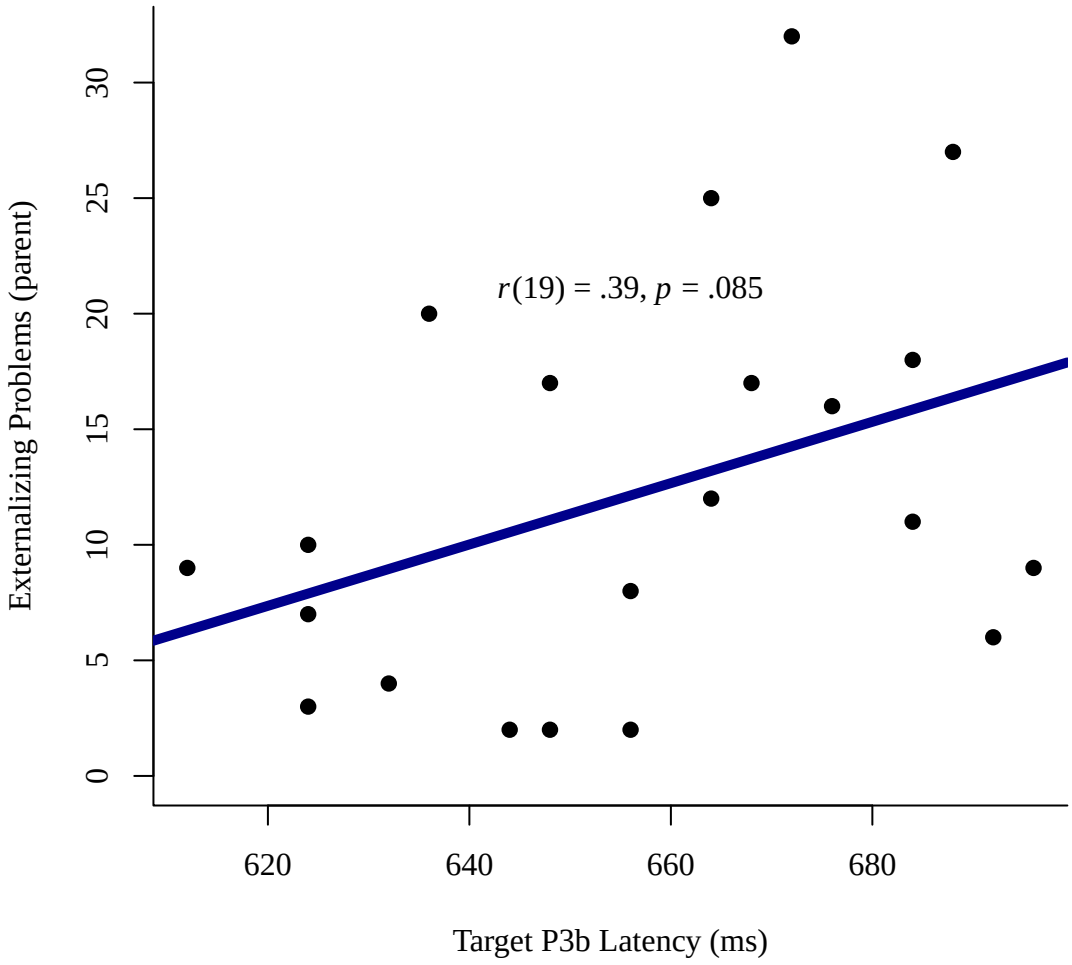

FIGURE 2.34: Association between target P3b latency and parent-reported externalizing problems on the CBCL.

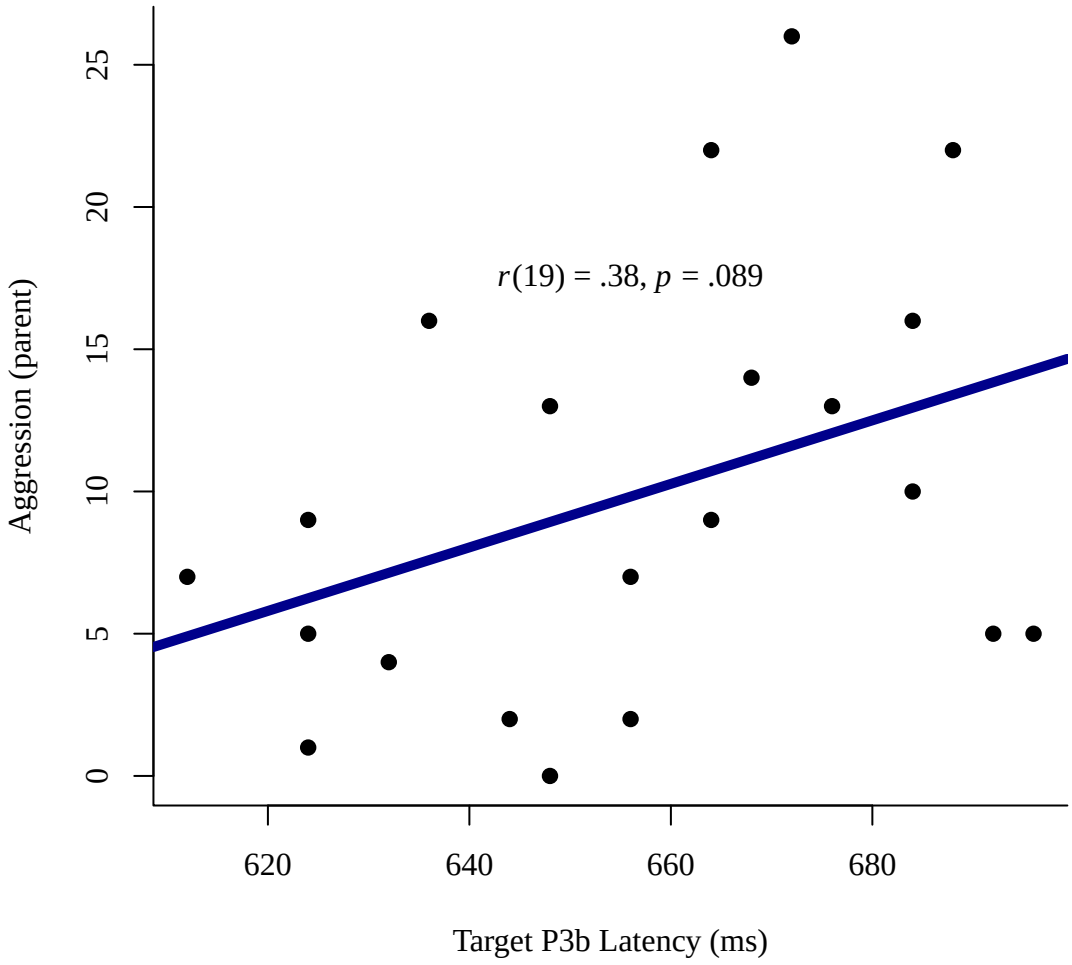

FIGURE 2.35: Association between target P3b latency and parent-reported aggression on the CBCL.

than linear fit in association with externalizing problems.

2.2.2.0.2 EEG and Externalizing Problems. Pearson correlations of children's EEG power values and asymmetry scores with their externalizing problems are in Table 2.19. Inconsistent with hypotheses, left frontal asymmetry (greater left than right frontal activation in the alpha frequency band) in the Bird/Alligator task was associated with *less* secondary caregiver-reported aggression.

Consistent with hypotheses, on the other hand, less frontal alpha power in both the oddball and Bird/Alligator tasks was associated with more behavior problems. Less frontal alpha power in the oddball task was associated with more parent-reported externalizing problems ($r[20] = -.60$, $p = .003$, see Figure 2.36), aggression ($r[20] = -.57$, $p = .005$, see Figure 2.37), and attention problems ($r[20] = -.55$, $p = .008$, see Figure 2.38). Associations of less frontal alpha power in the oddball task with externalizing problems ($p = .036$), aggression ($p = .040$), and attention problems ($p = .051$) remained significant when examining Spearman's rho. Clustered regression models examining the association of frontal alpha power with parent-reported externalizing problems, aggression, and attention problems are in Table 2.20. Associations of frontal alpha power in the oddball task with externalizing problems, aggression, and attention problems remained significant even after accounting for the nesting of longitudinal data and controlling for covariates.

Less frontal alpha power in the Bird/Alligator ERP task was marginally significantly associated with more parent-reported ECBI behavior problems ($r[20] = -.37$, $p = .091$, see Figure 2.41) and was significantly associated with more CBCL externalizing problems ($r[32] = -.50$, $p = .003$, see Figure 2.39) and aggression ($r[32] = -.52$, $p = .002$, see

Table 2.19: Study 1: Pearson Correlations of Children's EEG and Time-Frequency Components with their Externalizing Problems.

	Oddball			Bird/Alligator		
	Frontal Power	Frontal Asymmetry	Frontal TF	Frontal Power	Frontal Asymmetry	Frontal TF
CBCL EXT Parent	$-.60^{**}$	$-.21$	.29	$-.50^{**}$	.08	$-.03$
CBCL EXT Secondary	$-.05$	$-.60$	$-.54$	$-.31$	$-.71$	.41
CBCL AGG Parent	$-.57^{**}$	$-.20$	.31	$-.52^{**}$	.07	.03
CBCL AGG Secondary	$-.05$	$-.62$	$-.53$	$-.22$	$-.76^{\dagger}$	.32
CBCL ATT Parent	$-.55^{**}$	$-.24$	.12	$-.26$	.12	$-.26$
CBCL ATT Secondary	$-.01$	.18	$-.10$	$-.57$	.18	.60
ECBI Intensity Parent	$-.33$	.05	.34	$-.37^{\dagger}$	.26	$.38^{\dagger}$

Note. "TF" = time-frequency activity corresponding to timing of P3b (oddball) or N2 (Bird/Alligator, with values in decibels. "EXT" = externalizing problems, "AGG" = aggression, "ATT" = attention problems. Power values were log-transformed. Frontal power and asymmetry in alpha frequency range. Frontal time-frequency in theta frequency range. Frontal asymmetry reflects right frontal alpha power − left frontal alpha power (i.e., higher values reflect left frontal asymmetry in alpha frequency range). Correlations are two-tailed.

TABLE 2.20: Study 1: Clustered Regression Examining Association of Frontal Alpha Power (Oddball) with Parent-Reported CBCL Externalizing Problems, Aggression, and Attention Problems.

	Dependent variable:		
	CBCL Externalizing Problems (Parent)	CBCL Aggression (Parent)	CBCL Attention Problems (Parent)
Intercept	81.345^{***} (15.901)	57.394^{***} (14.657)	23.951^{***} (3.838)
Frontal Alpha Power (Log)	-18.852^{***} (1.605)	-15.739^{***} (1.431)	-3.113^{***} (0.392)
Sex	2.442 (1.818)	1.938 (1.609)	0.504 (0.519)
Age	1.103 (4.923)	4.313 (4.533)	-3.210^{***} (0.934)
Number of Bad Channels	1.000^{*} (0.469)	0.929^{*} (0.439)	0.071 (0.070)
Number of Target Trials Kept	-1.159^{***} (0.265)	-0.867^{***} (0.204)	-0.292^{***} (0.086)
Behavioral Percent Correct on Target Trials	0.204^{**} (0.066)	0.139^{*} (0.056)	0.065^{***} (0.015)
Observations	21	21	21
R^2	0.800	0.788	0.683
Adjusted R^2	0.714	0.697	0.547

Note. Age in years. Sex is coded as male = 0, female = 1. $^{\dagger}p < .10$. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

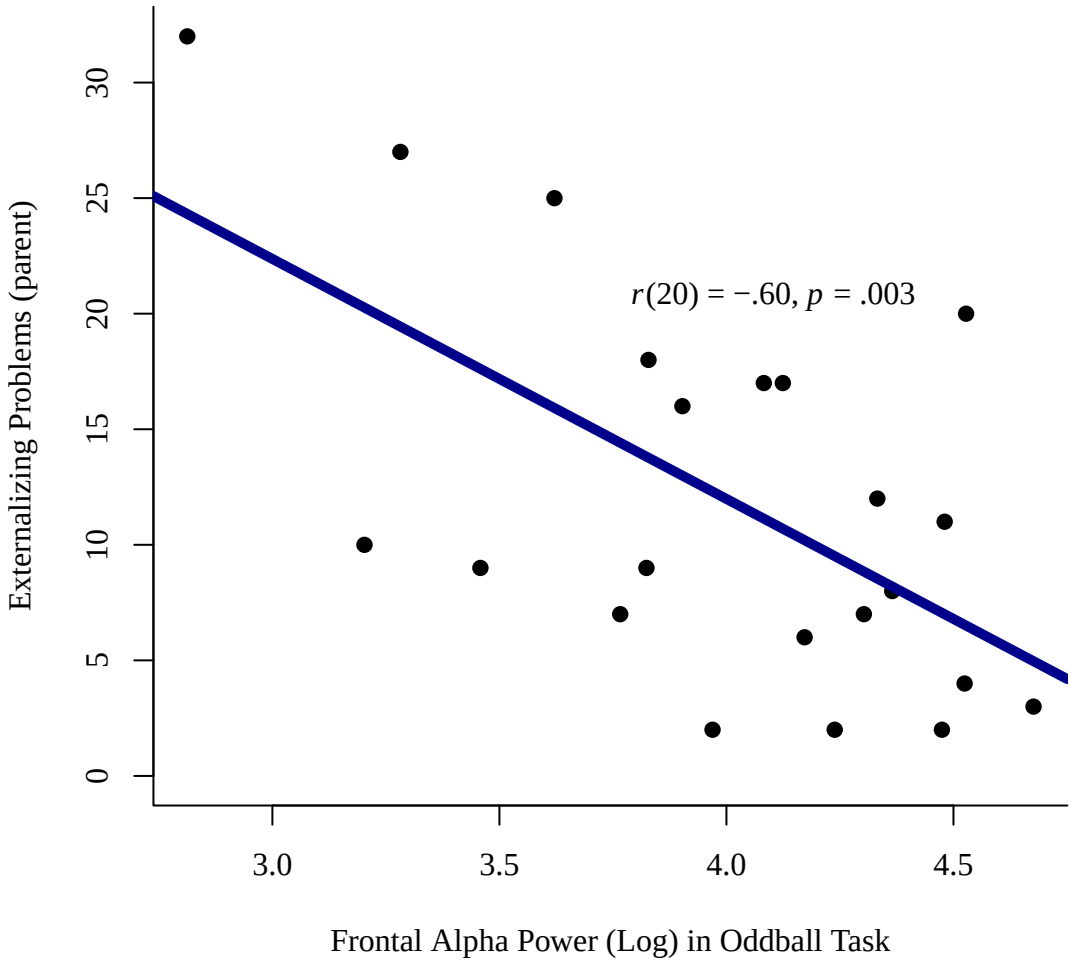

FIGURE 2.36: Association between frontal alpha power in the oddball task and parent-reported externalizing problems on the CBCL.

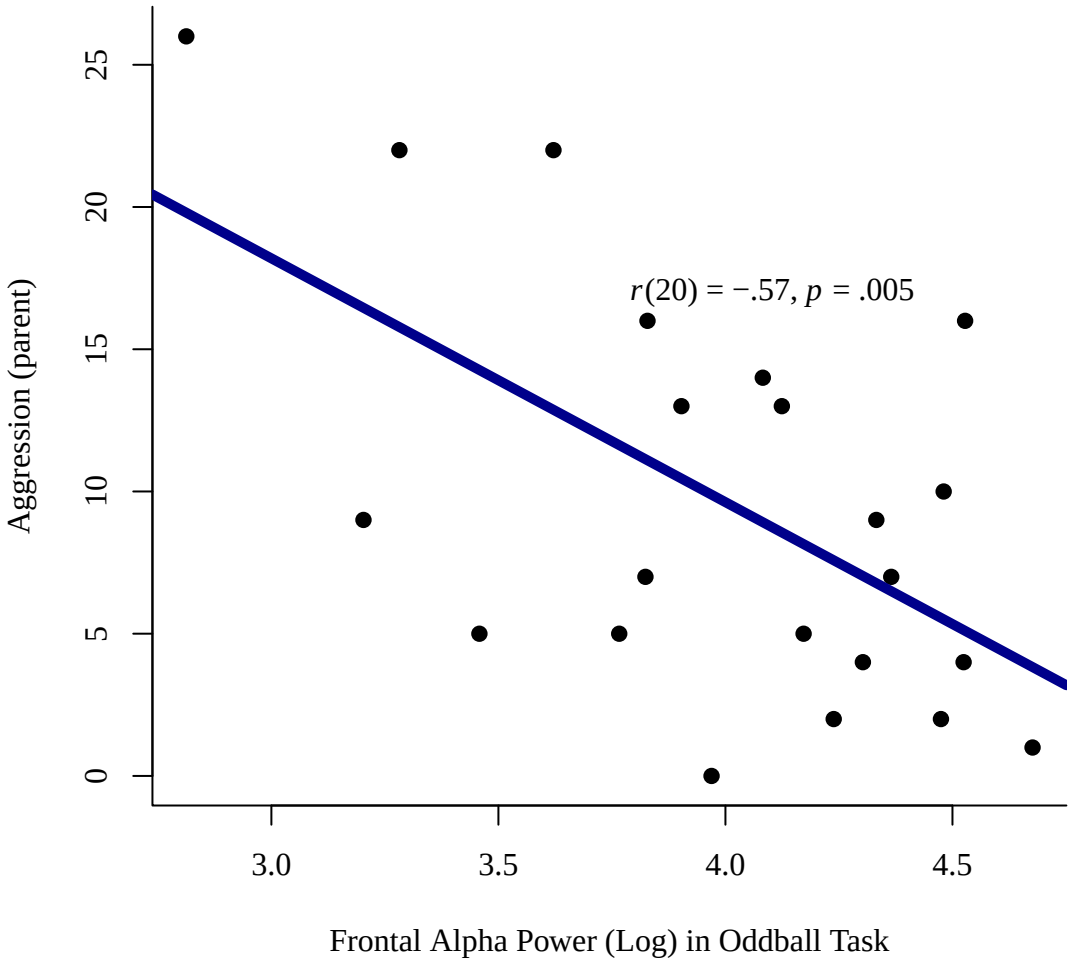

FIGURE 2.37: Association between frontal alpha power in the oddball task and parent-reported aggression on the CBCL.

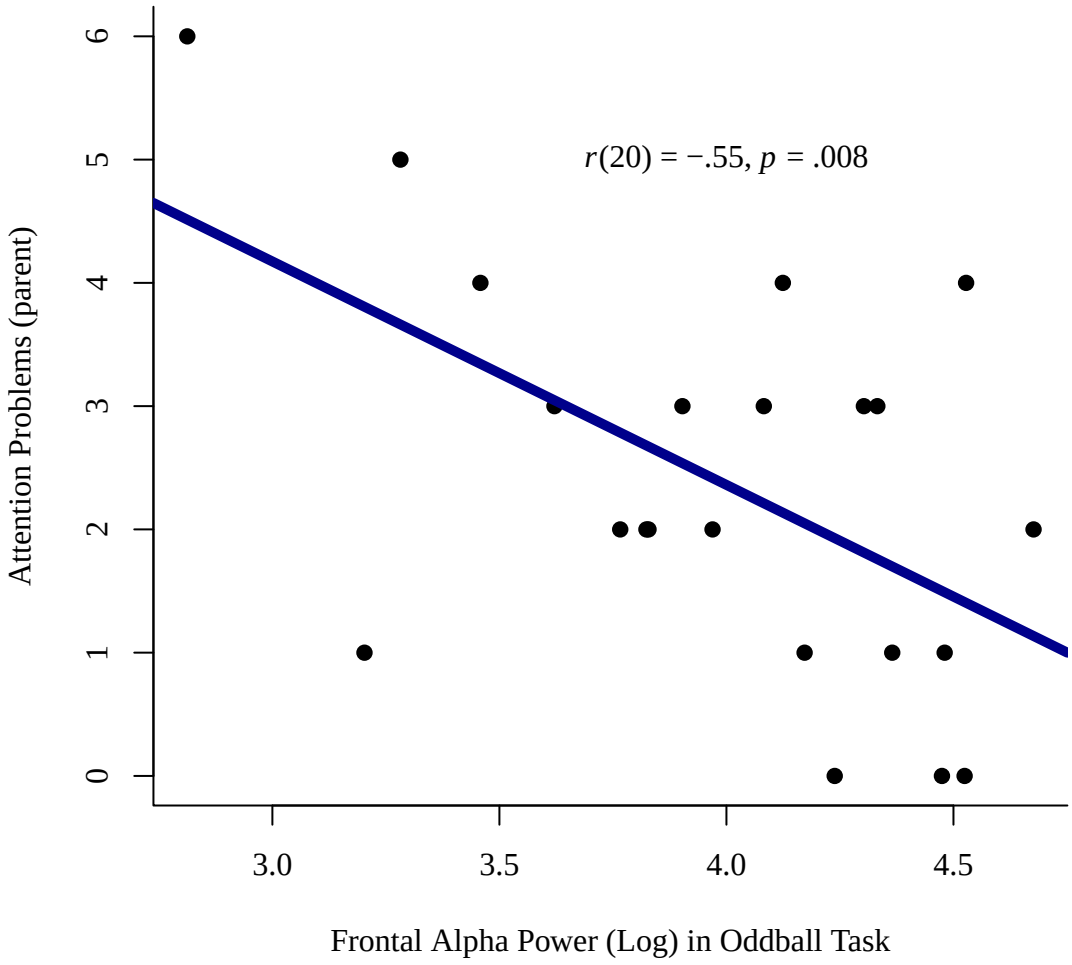

FIGURE 2.38: Association between frontal alpha power in the oddball task and parent-reported attention problems on the CBCL.

Figure 2.40). Associations of frontal alpha power in the Bird/Alligator task with CBCL externalizing problems ($p = .033$) and aggression ($p = .019$) remained statistically significant when examining Spearman's rho. However, associations of frontal alpha power in the Bird/Alligator task with ECBI behavior problems did not remain significant ($p = .268$), suggesting that the association between frontal alpha power and ECBI behavior problems may have owed, in part, to outliers. Clustered regression models examining the association of frontal alpha power with parent-reported externalizing problems and aggression are in Table 2.21. Associations of frontal alpha power in the Bird/Alligator task with CBCL externalizing problems and aggression remained significant even after accounting for the nesting of longitudinal data and controlling for covariates, whereas associations with ECBI behavior problems did not remain significant.

Because of the replicating finding across multiple tasks of less frontal alpha power in associations with behavior problems, we conducted follow-up analyses to examine the robustness of the association between frontal alpha power and behavior problems. We examined whether the effect was specific (a) to alpha power and (b) to frontal power. To examine whether the association was specific to alpha power, we examined whether frontal *theta* power (4–5 Hz) was associated with behavior problems. To examine whether the association was specific to frontal power, we examined whether alpha power at *posterior* electrodes was associated with behavior problems. Neither oddball ($r[20] = -.29$, $p = .195$) nor Bird/Alligator ($r[32] = -.14$, $p = .429$) frontal theta power was significantly associated with CBCL externalizing problems. Less posterior alpha power, on the other hand, was significantly associated with CBCL externalizing problems in both the oddball ($r[20] = -.60$, $p = .003$) and Bird/Alligator ($r[32] = -.60$, $p < .001$) ERP tasks.

TABLE 2.21: Study 1: Clustered Regression Examining Association of Frontal Alpha Power (Bird/Alligator) with Parent-Reported CBCL Externalizing Problems and Aggression and ECBI Behavior Problems.

	Dependent variable:		
	CBCL Externalizing Problems (Parent)	CBCL Aggression (Parent)	ECBI Behavior Problems (Parent)
Intercept	35.490* (14.662)	29.714* (11.592)	143.347** (52.240)
Frontal Alpha Power (Log)	−9.570** (3.013)	−8.685*** (2.387)	−26.575* (12.876)
Sex	3.017 (3.159)	2.423 (2.532)	18.132 (12.617)
Age	3.700 (3.970)	3.615 (3.365)	30.443 (19.770)
Number of Bad Channels	0.215 (0.262)	0.222 (0.205)	1.389 (1.301)
Number of No-Go Trials Kept	−0.194 (0.260)	−0.168 (0.217)	−1.369 (1.115)
Behavioral Percent Correct on No-Go Trials	0.012 (0.060)	0.016 (0.048)	−0.208 (0.209)
Observations	34	34	22
R^2	0.286	0.316	0.283
Adjusted R^2	0.127	0.164	-0.004

Note. Age in years. Sex is coded as male = 0, female = 1. $^{\dagger}p < .10$. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

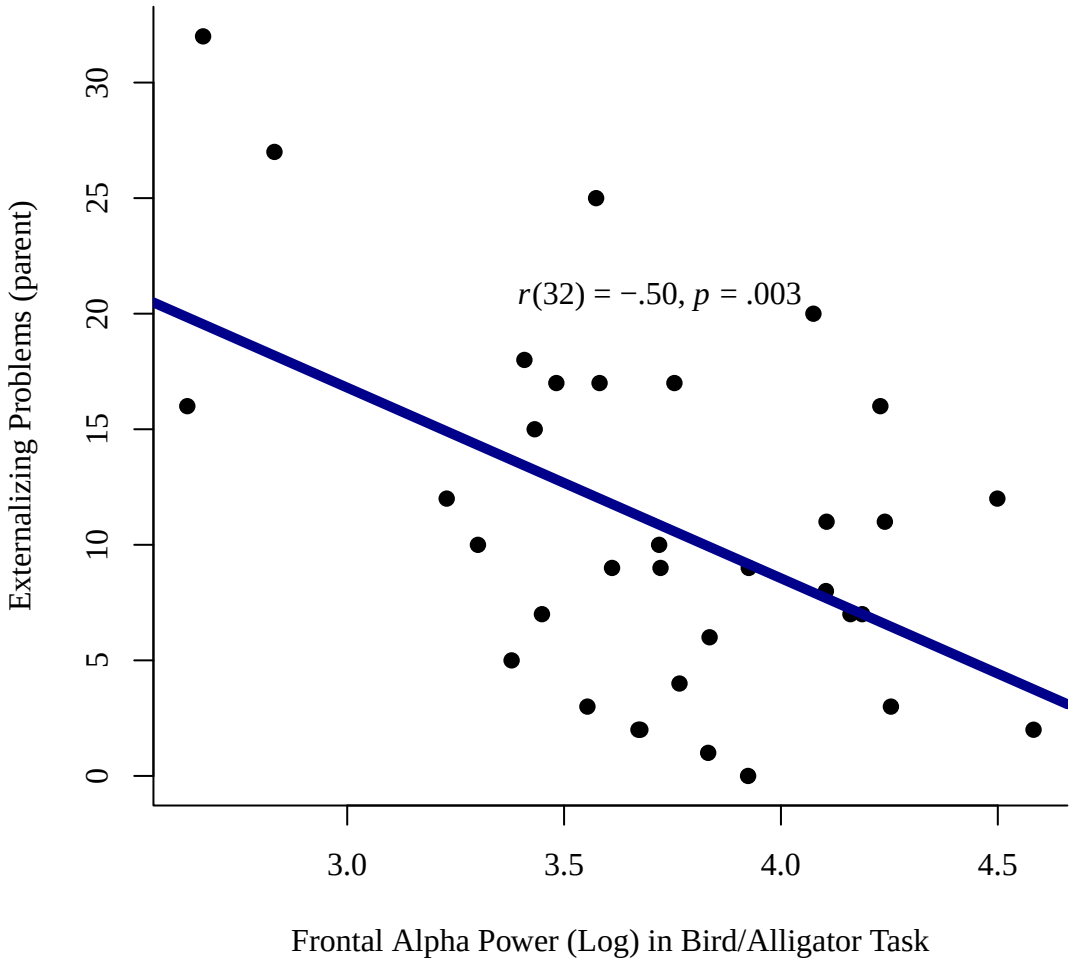

FIGURE 2.39: Association between frontal alpha power in the Bird/Alligator task and parent-reported externalizing problems on the CBCL.

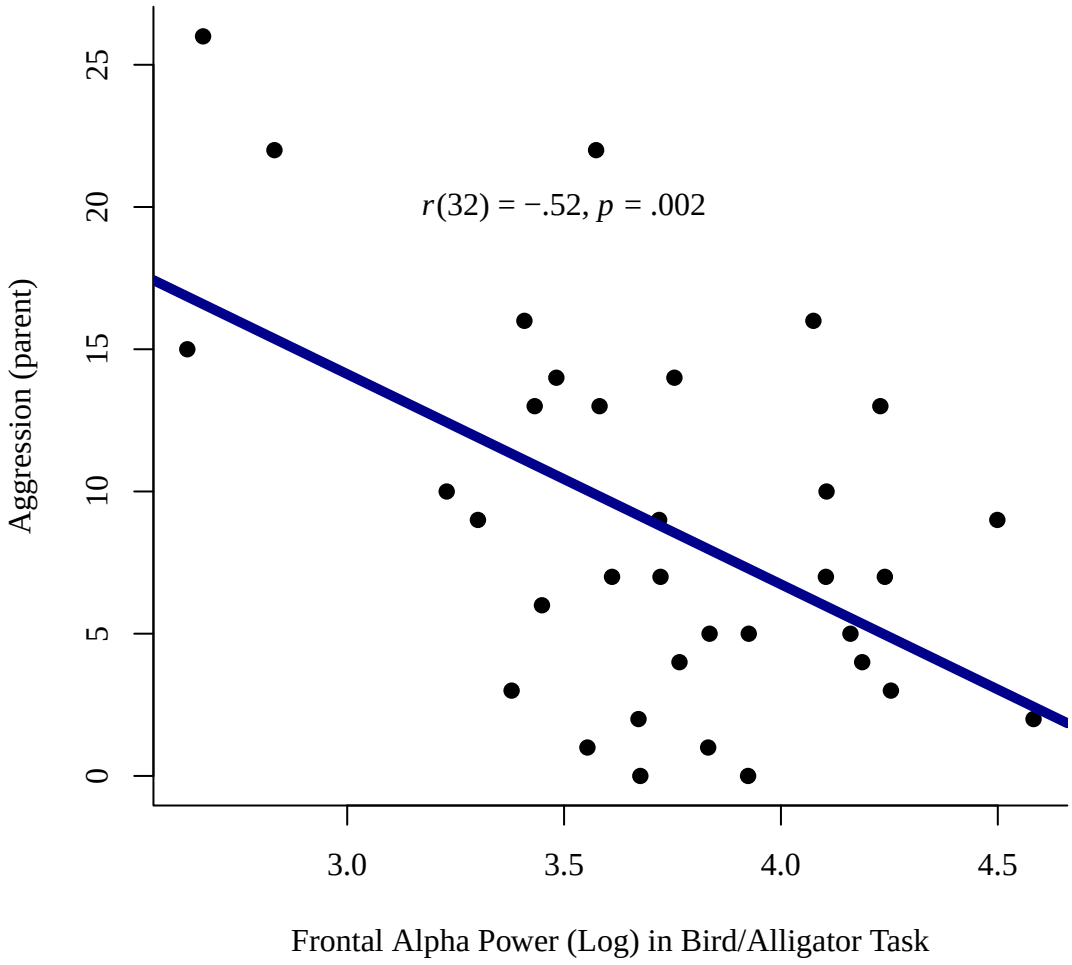

FIGURE 2.40: Association between frontal alpha power in the Bird/Alligator task and parent-reported aggression on the CBCL.

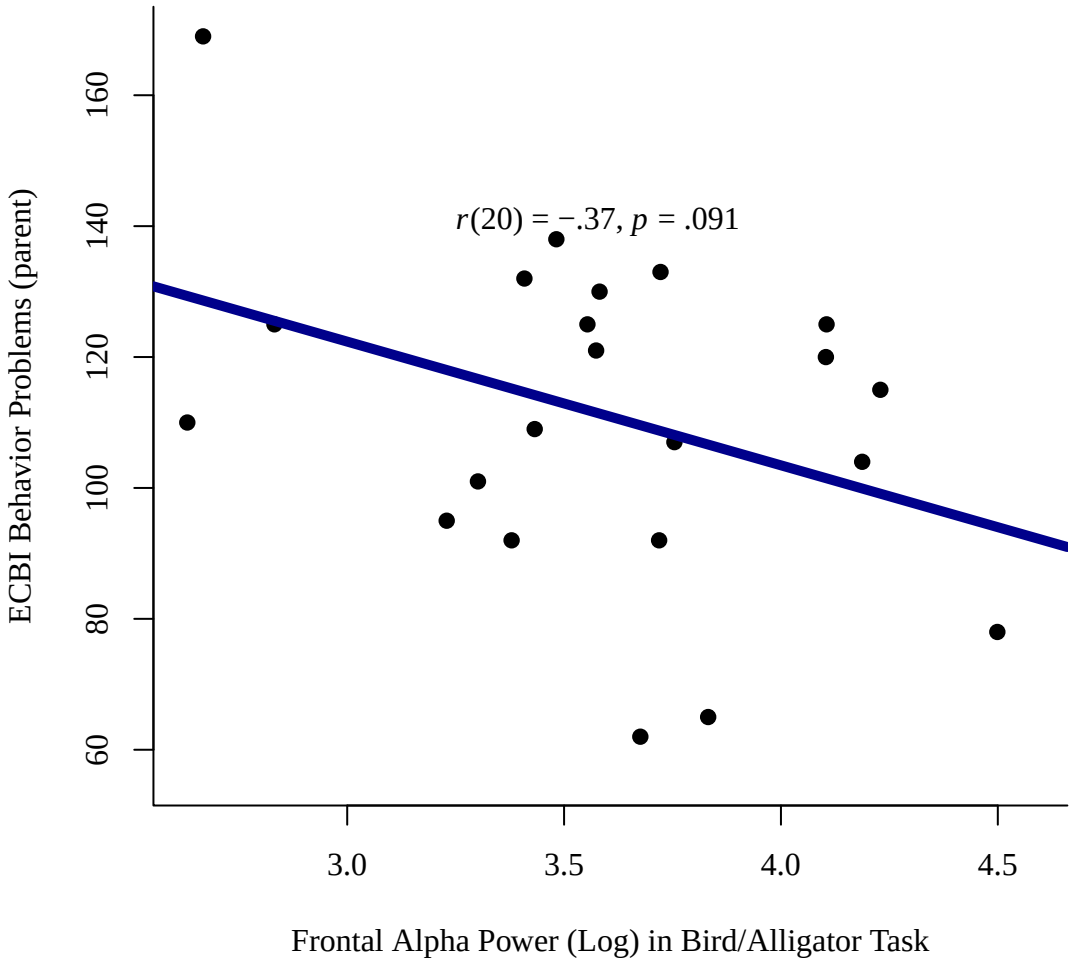

FIGURE 2.41: Association between frontal alpha power in the Bird/Alligator task and parent-reported intensity of behavior problems on the ECBI.

2.2.2.0.3 Time-Frequency Neurophysiology and Externalizing Problems.

Pearson correlations of children's time-frequency values and asymmetry scores with their externalizing problems are in Table 2.19. P3b-related frontal theta activity was not associated with externalizing problems. Inconsistent with hypotheses (but consistent with our associations between N2-related frontal theta activity and self-regulation), greater N2-related frontal theta activity was marginally significantly associated with greater intensity of behavior problems, as reported by parents on the ECBI ($r[20] = .38$, $p = .077$, see Figure 2.42). However, the association did not remain statistically significant at the .05 level when examining Spearman's rho ($p = .105$), suggesting that the association may have owed, in part, to outliers. A clustered regression model examining the association between N2-related frontal theta activity and parent-reported ECBI behavior problems is in Table 2.22. The association between N2-related frontal theta activity and ECBI behavior problems remained significant even after accounting for the nesting of longitudinal data and controlling for covariates.

Table 2.22: Study 1: Clustered Regression Examining Association Between N2-Related Frontal Theta Activity and ECBI Behavior Problems.

	Dependent variable:
	ECBI Behavior Problems (Parent)
Intercept	38.764 (50.960)
N2-Related Frontal Theta Activity	3.128* (1.446)
Sex	2.300 (13.826)
Age	33.697* (15.436)
Number of Bad Channels	−0.074 (1.014)
Number of No-Go Trials Kept	0.033 (0.671)
Behavioral Percent Correct on No-Go Trials	−0.253 (0.246)
Observations	22
R^2	0.310
Adjusted R^2	0.034

Note. Age in years. Sex is coded as male = 0, female = 1. $^{\dagger}p < .10$. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

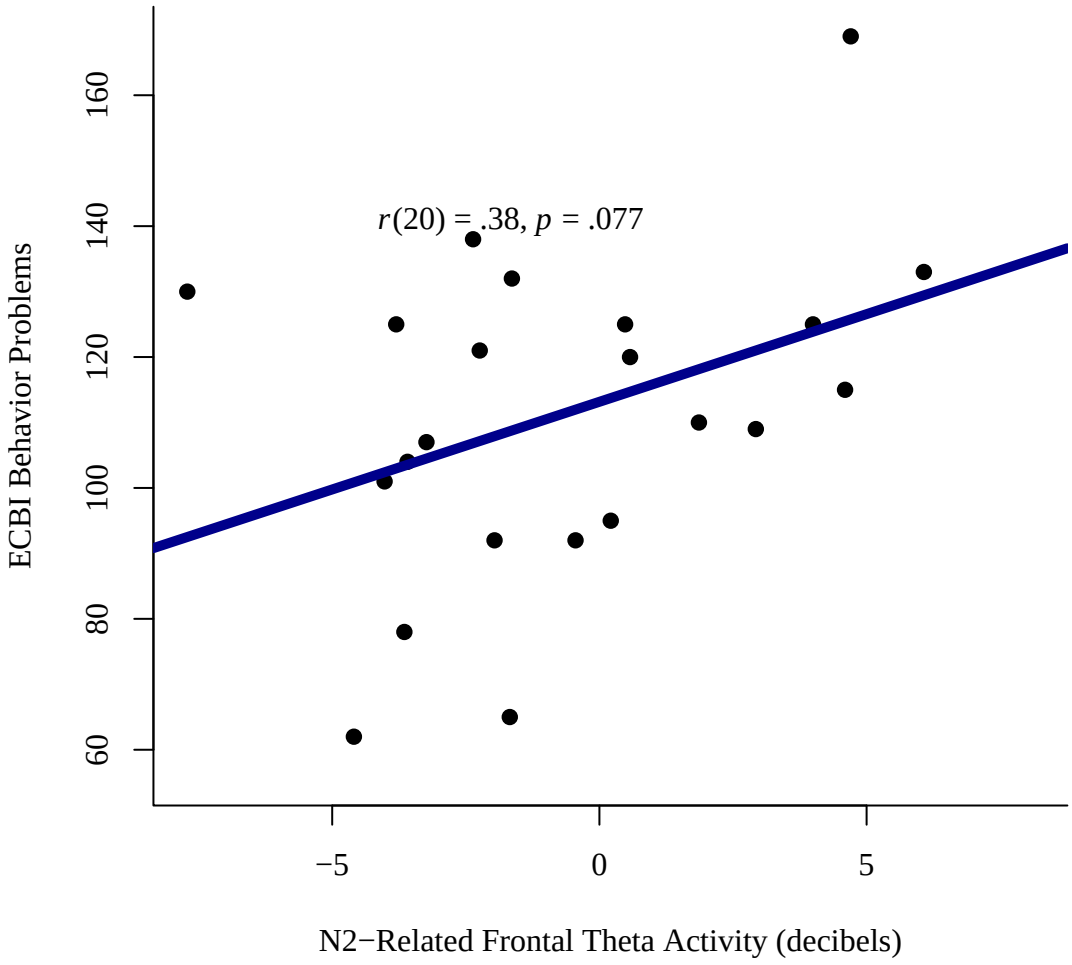

FIGURE 2.42: Association between N2-related frontal theta activity in the Bird/Alligator task and parent-reported intensity of behavior problems on the ECBI.

2.2.3 Association Between Self-Regulation and Externalizing Problems

For more accurate estimates of the association between self-regulation and behavior problems, we used the full sample of 336 families who were part of the larger study (i.e., not just those who were recruited for the EEG procedures). Relatively few concurrent associations were observed between self-regulation and externalizing problems (see Table 2.23). Those self-regulation variables that were associated with behavior problems (in ways that were consistent with hypotheses) were primarily observed with secondary caregivers' rather than parents' reports of behavior problems. For instance, better performance on Token Sort was associated with fewer attention ($r[170] = -.25$, $p < .001$) and externalizing ($r[170] = -.18$, $p = .019$) problems and, at a trend level, with less aggression ($r[170] = -.14$, $p = .073$), as reported by secondary caregivers. In addition, better performance on the Bird/Alligator inhibitory control task was associated with fewer externalizing problems ($r[203] = -.11$, $p = .111$) and, at a trend level, with fewer attention problems ($r[203] = -.15$, $p = .031$) as reported by secondary caregivers, but only with (fewer) attention problems among parents' reports ($r[463] = -.13$, $p = .007$). A few associations were observed that were inconsistent with hypotheses. Better performance on Token Sort was associated with *more* parent-reported behavior problems on the ECBI and, at a trend level, with more parent-reported aggression. Also, better performance on the target trials of the oddball ERP task was associated with more parent-reported externalizing problems, but this was based on data from the smaller ERP sample.

Table 2.23: All Studies: Pearson Correlations of Children's Self-Regulation with Ratings of Their Externalizing Problems.

	CBCL EXT	CBCL EXT (Sec)	CBCL AGG	CBCL AGG (Sec)	CBCL ATT	CBCL ATT (Sec)	ECBI
Bird/Alligator	−.08	−.11	−.05	−.09	−.13**	−.15*	−.06
Shape Stroop	−.03	−.01	−.02	.00	−.04	−.03	.03
Grass/Snow	.02	−.01	.02	.00	.03	−.05	.01
Token Sort	.07	−.18*	.09†	−.14†	.00	−.25**	.12**
Sustained Play Attention	−.02	−.05	−.03	−.04	.02	−.06	−.01
Bird/Alligator ERP	−.10	−.13	−.08	−.02	−.14	−.69	−.26
Oddball	.31†	.04	.30†	.11	.28	−.39	.05

Note. $^{\dagger}p < .10$. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$. "EXT" = Externalizing Problems. "AGG" = Aggression. "ATT" = Attention Problems. "Sec" = secondary caregiver-reported. "All Studies" refers to the full sample of 336 families who were part of the larger study (i.e., not just those who were recruited for the EEG procedures). Correlations are two-tailed.

2.2.4 Mediation

Based on the patterns of associations, we tested two possible mediational processes:

1. Smaller target P3b amplitude → poorer inhibitory control in the Bird/Alligator task → more behavior problems

2. Less N2-related frontal theta activity → poorer inhibitory control in the Bird/Alligator task → more behavior problems

Performance on the Bird/Alligator inhibitory control task was associated with secondary caregiver-reported externalizing problems, secondary caregiver-reported attention problems, and parent-reported attention problems, so we examined these three measures of behavior problems in the mediation models. We note, however, that sample sizes were especially small when examining secondary caregiver-reported behavior problems, so we present the mediational results with caution. The confidence interval included zero when examining the indirect effect of target P3b amplitude on parent- (−0.04, 0.05) and secondary caregiver- (−0.20, 0.16) reported attention problems via performance on Bird/Alligator. On the other hand, the confidence interval of the indirect effect of target P3b amplitude on secondary caregiver-reported externalizing problems via performance on Bird/Alligator did not include zero (0.04, 1.06), suggesting a significant mediated effect. A path diagram of the model is in Figure 2.43. Larger target P3b amplitudes were associated with better performance on Bird/Alligator. However, performance on Bird/Alligator was not significantly associated with secondary caregiver-reported externalizing problems in the model (although the coefficient was in the opposite direction as hypothesized). Target P3b amplitudes remained

associated with externalizing problems even when controlling for performance on Bird/Alligator, suggesting that the indirect effect involved partial rather than full mediation. The significant indirect effect suggested that children with smaller P3b amplitudes were reported to have more externalizing problems, in part, because they had poorer inhibitory control. The effect size of the mediation effect was calculated as the ratio of the indirect effect over the total effect from P3b amplitudes to externalizing problems, which represents the proportion of effect mediated (P_M; Shrout & Bolger, 2002). The P_M was .49, indicating that poorer inhibitory control accounted for nearly half of the association between smaller P3b amplitudes and more externalizing problems. Data were cross-sectional, however, so we were unable to test the possibility of other directions of effect.

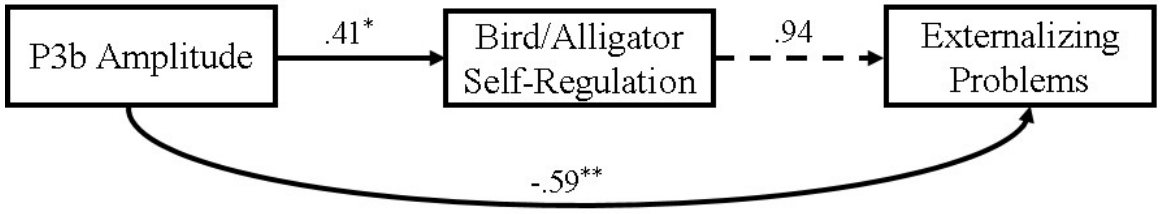

FIGURE 2.43: Indirect effect of target P3b amplitude on secondary caregiver-reported externalizing problems via performance on the Bird/Alligator inhibitory control task. Coefficients are standardized regression coefficients.

We then examined whether N2-related frontal theta activity had an indirect effect on behavior problems via performance on the Bird/Alligator inhibitory control task. The confidence interval included zero when examining whether performance on Bird/Alligator mediated the effect of N2-related frontal theta activity on parent- (−0.10, 0.07) and secondary caregiver- (−0.49, 0.42) reported attention problems and secondary caregiver-reported externalizing problems (−3.41, 0.40). Thus, inhibitory control on Bird/Alligator did not mediate the effect of N2-related frontal theta activity on behavior problems.

2.2.5 Effect Sizes

Given the relatively small sample size and the resulting modest power to detect associations, we were only able to detect associations of fairly large magnitude with neurophysiological variables. Nevertheless, there were some replicating patterns of effects. Smaller target P3b amplitudes and smaller P3b amplitude difference scores were associated with poorer self-regulation in the Bird/Alligator inhibitory control task ($|r| \approx .4$) and more secondary caregiver-rated externalizing problems ($|r| \approx .8$) with medium-to-large effect sizes. Moreover, less frontal alpha power in both the oddball and Bird/Alligator tasks was associated with more parent-rated externalizing problems and aggression (less oddball frontal alpha power was also associated with attention problems). Additionally, less frontal alpha power in the Bird/Alligator task was associated with worse behavioral performance on the same task. Associations of frontal alpha power with self-regulation ($|r| \approx .3$) and externalizing problems ($|r| \approx .6$) had medium-to-large effect sizes. Also, there were relatively few effects that were in the opposite direction from what we hypothesized (except N2-related frontal theta activity), suggesting that many of the observed effects were likely not spurious. Although N2-related frontal theta activity was associated with self-regulation and externalizing problems in ways that were inconsistent with hypotheses, it showed a consistent pattern. Greater N2-related frontal theta activity was associated with poorer self-regulation in the oddball and Bird/Alligator inhibitory control tasks and more parent-reported externalizing problems. Associations between N2-related frontal theta activity and self-regulation/externalizing problems had medium effect sizes ($|r| \approx .3$ to $.4$). Associations between self-regulation and externalizing problems were based on a larger sample, so we

had greater power to detect effects. Most observed associations between self-regulation and externalizing problems had small-to-medium effect sizes ($|r| \approx .1$ to .3).

2.3 Discussion

Study 1 examined associations between neural functioning, self-regulation, and externalizing behavior. There was some evidence that neural functioning predicted self-regulation. Consistent with hypotheses, smaller target P3b amplitudes and N2/P3b amplitude difference scores were associated with poorer self-regulation. Smaller target P3b amplitudes and P3b amplitude difference scores were associated with worse performance on Bird/Alligator inhibitory control. Smaller N2 amplitude difference scores were associated with worse performance on Shape Stroop. Also consistent with hypotheses, less frontal alpha power, left frontal asymmetry, and less N2-related frontal theta activity were associated with poorer self-regulation. Less frontal alpha power in the Bird/Alligator task was associated with worse performance on the same task. Left frontal asymmetry was associated with worse performance on Shape Stroop. Inconsistent with hypotheses, however, longer target P3b latencies were associated with better performance on Grass/Snow, left frontal asymmetry was associated with better performance on the Sustained Play Attention task, and more N2-related frontal theta activity was associated with worse behavioral performance on the oddball ERP and Bird/Alligator inhibitory control tasks.

There was also evidence of neural functioning predicting externalizing problems. Consistent with hypotheses, smaller target P3b amplitudes, smaller P3b amplitude difference scores, and longer target P3b latencies were associated with more externalizing problems.

Smaller target P3b amplitudes and smaller P3b amplitude difference scores were associated with more externalizing problems, as rated by secondary caregivers. Longer target P3b latencies were associated with more externalizing problems and aggression, as rated by parents. Inconsistent with hypotheses, however, left frontal asymmetry was associated with fewer secondary caregiver-reported externalizing and aggression problems, and more N2-related frontal theta activity was associated with more parent-reported externalizing problems.

Consistent with hypotheses, on the other hand, less frontal alpha power was associated with more externalizing problems, aggression, and attention problems. Less frontal alpha power in the oddball task was associated with more externalizing problems, attention problems, and aggression, as rated by parents. Less frontal alpha power in the Bird/Alligator task was associated with more externalizing problems and aggression, as rated by parents. Findings of less frontal alpha power in externalizing problems were fairly specific to alpha power (as opposed to theta power) but were not specific to frontal power—similar associations were observed with posterior alpha power.

There was some evidence that self-regulation mediated the effects of neural functioning on externalizing problems. Consistent with hypotheses, poorer inhibitory control on Bird/Alligator partially mediated the effect of smaller target P3b amplitudes on secondary caregiver-reported externalizing problems. However, no other mediational effects were observed.

In summary, there was no evidence in support of Hypothesis 1 that externalizing problems would be associated with smaller amplitudes of the no-go N2. There was also no

evidence for Hypothesis 2 that externalizing problems would be associated with longer latencies of the no-go N2. On the other hand, there was some support for Hypotheses 3 and 4 that externalizing problems would be associated with smaller amplitudes and longer latencies of the oddball P3b. There was no support for Hypothesis 5 that externalizing problems would be associated with left frontal asymmetry in the alpha frequency band. There was fairly robust support for Hypothesis 6 that externalizing problems would be associated with less frontal alpha power. There was no support for Hypothesis 7 that externalizing problems would be associated with less N2-related frontal theta activity (it was in the opposite direction as hypothesized) and P3b-related frontal theta activity. There was no support for Hypotheses 8 and 9 that the no-go N2 would be associated with disinhibition whereas the P3b would be associated with sustained attention. Finally, there was a little support for Hypothesis 10 that self-regulation would mediate the association between neural functioning and externalizing problems.

The lack of stronger evidence supporting a mediational model might reflect the cross-sectional nature of the data. A causal chain of events may take time to unfold, so cross-sectional data may be insufficient for examining the mediational questions of interest in the present study. Cross-sectional models can be biased for mediation tests because assumptions of stationarity (i.e., constant relations among variables over time) are unlikely to be met (Maxwell & Cole, 2007; Maxwell, Cole, & Mitchell, 2011). Study 2 includes longitudinal data that permit a stronger, more powered test of mediation.

Chapter 3

Study 2

3.1 Method

3.1.1 Participants

Children and their families were recruited in the period October 2013 to March 2015 from the Bloomington, Indiana area to participate in a study with assessments of neural functioning, self-regulation, and behavior problems at three ages: 30, 36, and 42 months. All assessments were conducted within two weeks of the child's target age. Participants were recruited in similar ways to Study 1. Of the 111 families, 89 (80%) agreed to participate in the EEG procedures. Of those who agreed to participate in the EEG procedures, 75 (84%) had an EEG visit. Children were assessed with EEG procedures at 30 (n = 49), 36 (n = 28), and 42 (n = 27) months of age (this includes some children who were assessed at multiple occasions). Children were included in the analyses for the present report if they had usable EEG data at one or more measurement occasions, resulting in a final sample of 64 unique children. In order to examine the co-development of brain functioning and behavior over time, some children (n = 20) had multiple EEG assessments, resulting in a final sample of

$N = 87$ cases. Mean age at the EEG assessment was 36.52 months ($SD = 5.34$). Of the final sample, 25 (39%) children were girls, and 39 (61%) were boys. Although it would have been ideal to have longitudinal data on all children, using the available longitudinal data provides better power than using only one time point (and discarding the remaining data), given that we can account for dependency in the data.

Parents (usually the mother) reported on the child's behavior problems. Among the parents, 61 (95%) were female, 95% were Caucasian, 3% were Hispanic, and 2% were African-American. Information on child ethnicity was not collected. There were 61 mothers, 3 fathers, and 97% were biological parents. Parents ranged in age from 24 to 53 years old ($M = 33.82$, $SD = 4.84$). Of parents, 93% were married, 5% were single, and 2% were divorced. Among parents, 86% had a college degree, 10% had completed some college, 3% had a high school diploma, and 1% had completed 8th grade or less. The Hollingshead four-factor index of SES (Hollingshead, 1975) ranged from 13 to 66 ($M = 46.99$, $SD = 14.09$), suggesting a sample with some variation in SES, but with a solid middle-class core.

In addition to collecting parent reports of behavior problems, with the parents' permission, we asked secondary caregivers to rate behavior problems. Secondary caregivers were persons (over age 18) not living with the child who spent the most time with the child in the past 30 days. To collect additional secondary caregivers, we removed the inclusion criterion from Study 1 that secondary caregivers had to spend at least 10 hours with the child in the past 30 days. Parents did not name a secondary caregiver at ages 30, 36, and 42 months for 36%, 32%, and 33% of the children, respectively. Of the children whose parents named a secondary caregiver, 48%, 47%, and 50% of their secondary caregivers participated at 30, 36, and 42 months, for a total of 46 secondary caregivers. Of these secondary caregivers,

67% were teachers, 26% were other relatives, 5% were babysitters, and 2% had other connections to the child. Secondary caregivers spent, on average, 11.59 hours (SD = 4.87) with the target child in the month prior to assessment.

3.1.2 Measures

3.1.2.1 Neurophysiology

3.1.2.1.1 Electrophysiological Data Acquisition. The electrophysiological data acquisition procedures were similar to those in Study 1 (see Section 2.1.2.1.1).

3.1.2.1.2 Electrophysiological Tasks. 1. P3a oddball task, 6 minutes: an auditory oddball (two-tone discrimination) task was used to elicit a P3a (P300) ERP component to infrequent sounds. Sounds consisted of pure, low-frequency (1000 Hz) and high-frequency (1500 Hz) tones. Sounds denoting the infrequent stimulus were counterbalanced across children. One hundred twenty sounds lasting 1200 ms occurred with an interstimulus interval of 1400–1600 ms. Sounds were randomly ordered so that one occurred 70% of the time and the other occurred on 30% of the trials, for a total of 84 frequent and 36 infrequent (target) trials. Children were not asked to make a behavioral response. To retain similar numbers of trials per condition and ensure similar signal-to-noise ratios across conditions, only frequent trials directly preceding target trials were kept. This was not done in the other electrophysiological tasks because they involved a behavioral response and, as a result, fewer trials. Participants had, on average, 9.00 (SD = 3.06) bad electrode channels during the task. Participants contributed 18.99 (SD = 4.67) usable target and 19.42 (SD = 5.67) usable frequent trials on average.

2. Inhibitory control task, 6 minutes: a go/no-go task, in which the child was asked to press a button to catch the fish (see Figure 3.1) in the child's net and not to catch the sharks (see Figure 3.2), was used to elicit a response inhibition potential (N2 or N200) on the no-go trials. The *Fish/Sharks* task was designed for use with young children (Wiebe, Sheffield, & Espy, 2012) and was adapted for use with ERP measures (Wiebe, Carroll, Raber, and Espy, 2007; adapted from Simpson and Riggs, 2006). Children were instructed to push the large green button when they see a fish but not when they see a shark. Following a button press, the child was given feedback indicating whether the response was accurate. On correct button presses when the fish was displayed, a picture of the net with the fish caught in the net was displayed (see Figure 3.3), along with a bubbling sound indicating the fish was caught. On incorrect button presses when the shark was displayed, a picture of the net with the shark breaking the net was displayed (see Figure 3.4), along with a buzzer sound. Four sets of 4 practice trials were administered in the following order: go trials (fish only), no-go trials (sharks only), go trials, and mixed go- and no-go trials (fish and sharks trials intermixed). Children were trained to 75% criterion in each practice session. Following successful completion of practice trials, the test trials were presented. Stimuli were presented randomly so that a fish appeared on 75% of trials and a shark on the other 25%. During picture presentation, children's ERPs were recorded to the appearance of the picture of the fish or shark for 3000 ms, or until the child pressed the button. If the child pressed the button, feedback was presented for 750 ms after an 800 ms delay following the button press. The interstimulus interval was 1500 ms between the end of the previous trial stimulus or feedback and the onset of the next trial stimulus. There were a total of 80 trials, with 60 go and 20 no-go trials. Trials were block-randomized so that each block of

eight trials included six fish trials and two shark trials: one shark trial followed two fish trials, and the other shark trial followed four fish trials. All exemplars (10 fish, 3 sharks) appeared with roughly equivalent frequency. Trials with responses faster than 200 ms were rejected because they were too quick to reflect a response to the current stimulus.

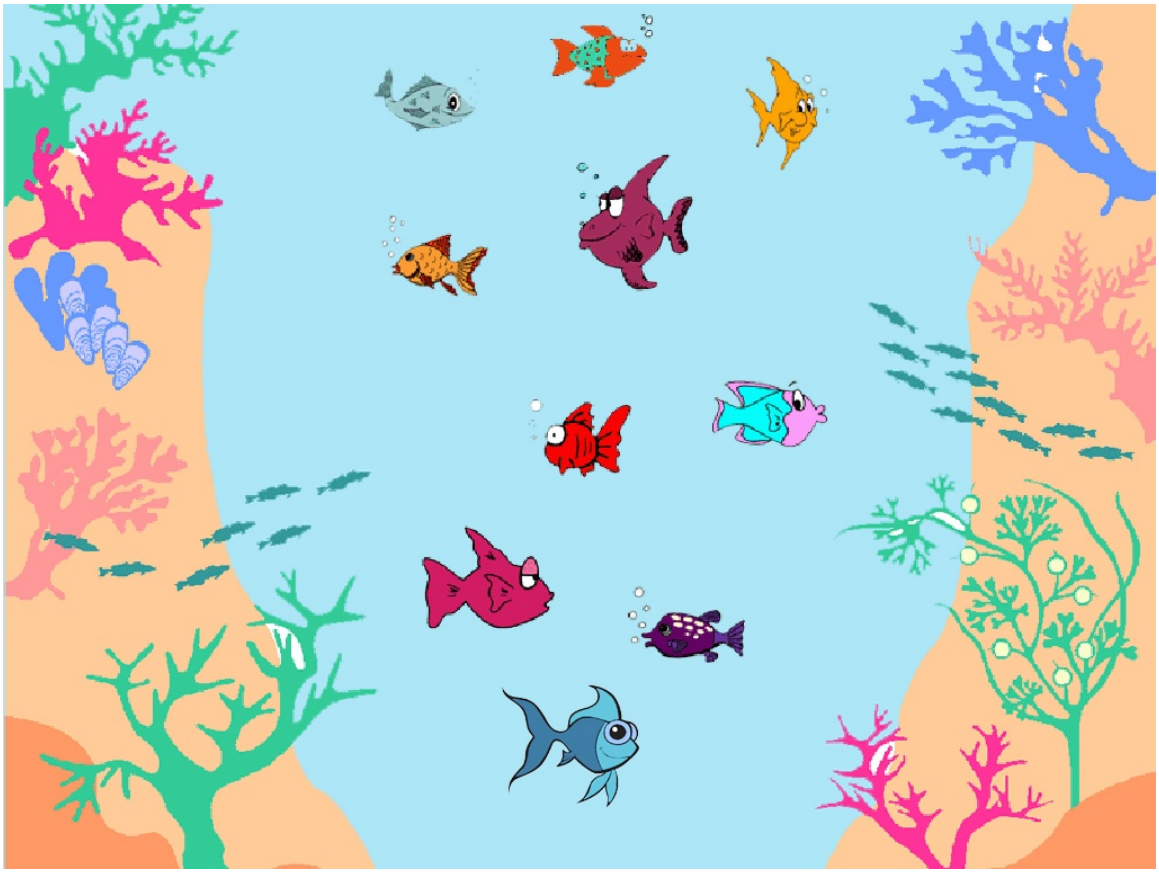

FIGURE 3.1: Picture of fish exemplars in Fish/Sharks task.

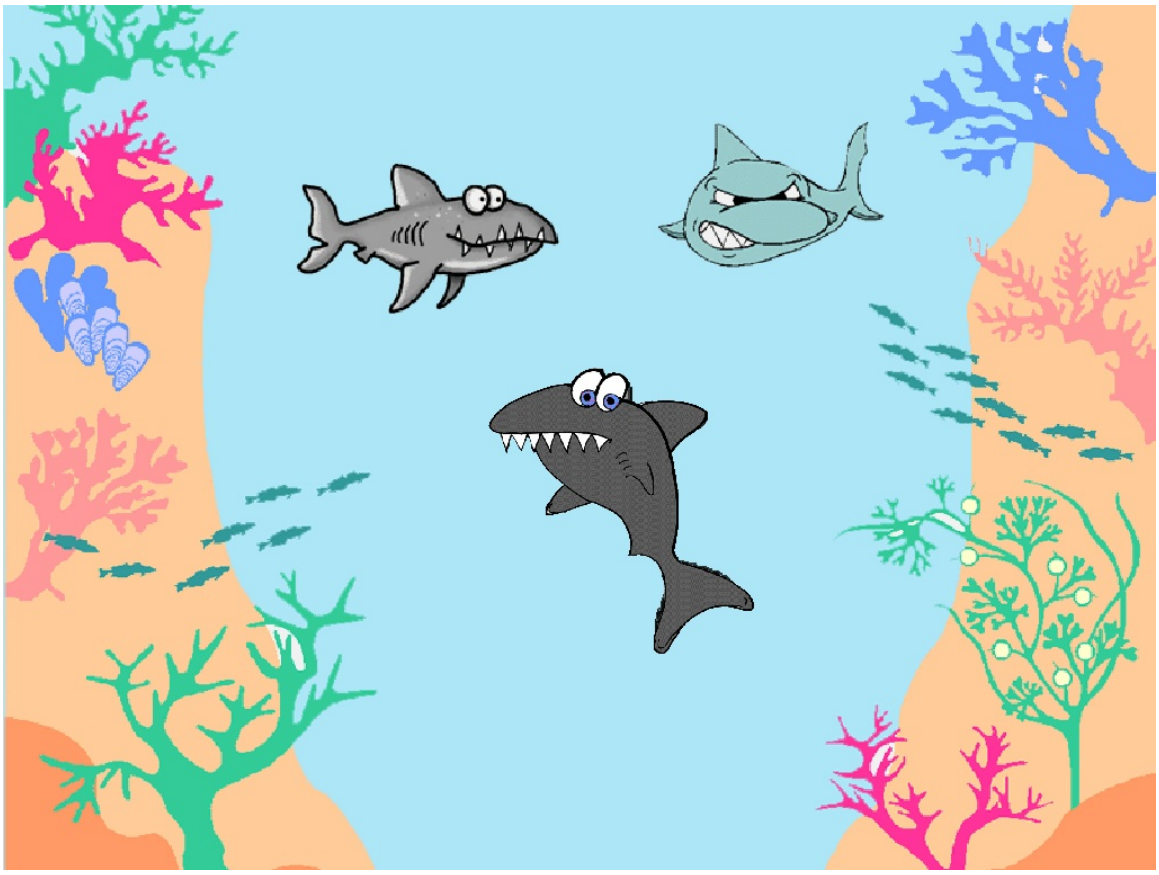

FIGURE 3.2: Picture of shark exemplars in Fish/Sharks task.

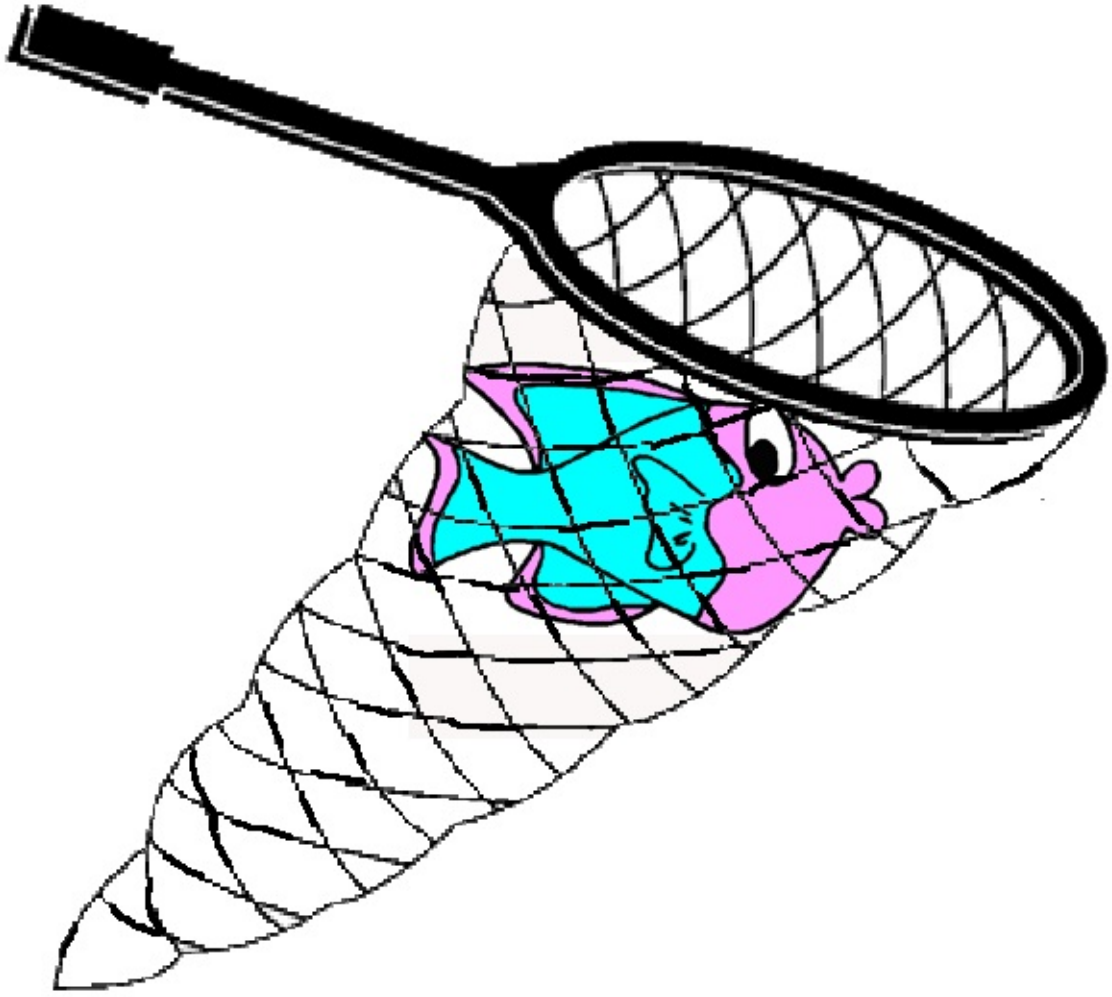

FIGURE 3.3: Picture of feedback on go (fish trials) in Fish/Sharks task.

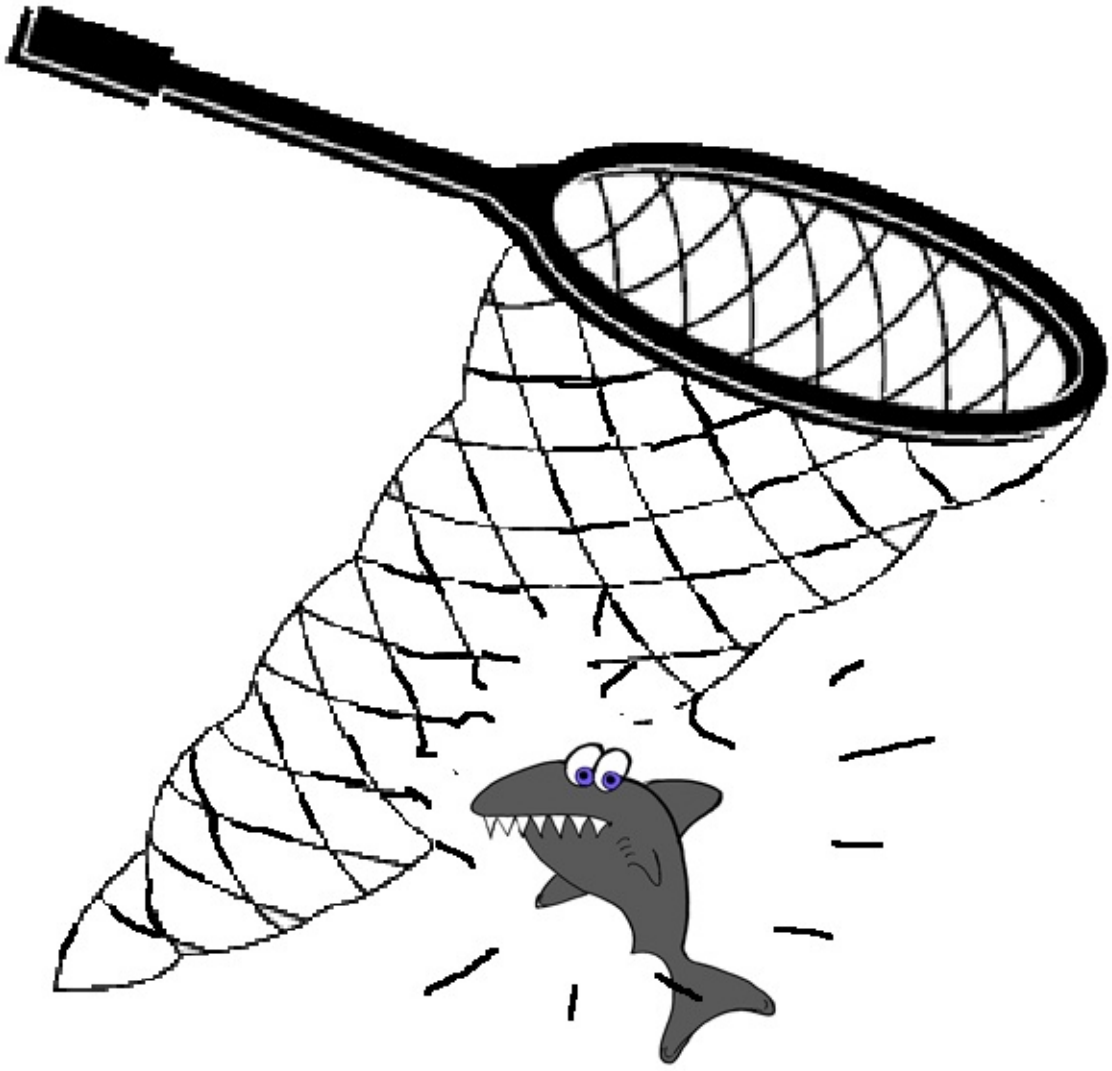

FIGURE 3.4: Picture of feedback on no-go (shark trials) in Fish/Sharks task.

Participants had, on average, 11.28 ($SD = 3.50$) bad electrode channels during the task. Participants contributed 27.39 ($SD = 17.04$) usable go and 10.32 ($SD = 5.92$) usable no-go trials on average. Means and 95% CIs of behavioral performance (percent correct) on each trial condition are presented in Figure 3.5. Children responded more frequently to the relevant go stimuli than the no-go stimuli ($t[80] = 10.43$, $p < .001$), suggesting that children successfully categorized the stimuli and were fairly successful at selectively responding to the go stimuli.

There were offsets between E-Prime's command to present the stimuli and the actual presentation of the stimuli in both tasks. Offsets were approximately 4 ms, on average, for the auditory tones in the oddball task. Offsets were approximately 50 ms, on average, for the visual fish stimuli and 51 ms, on average, for the visual shark stimuli in the Fish/Sharks task. There were also analog-to-digital conversion delays of 8 ms for the EGI Net Amps 300 series amplifier. These offsets and delays were accounted for by shifting the windowed time frame later during trial segmentation by the sum of the offset and the 8 ms analog-to-digital conversion delay.

3.1.2.1.3 ERP Data Processing. The processing of ERP data was similar to the data processing in Study 1 (see Section 2.1.2.1.3). Unlike in Study 1, however, only correct trials were used in the calculation of participants' average waveforms. We only included correct trials in Study 2 because the tasks were more child-friendly (the go/no-go task involved performance feedback, and the oddball task did not require a behavioral response). Including only correct trials in Study 2 also allowed us to see whether we could replicate findings from Study 1, which might inform us of the degree to which findings might be

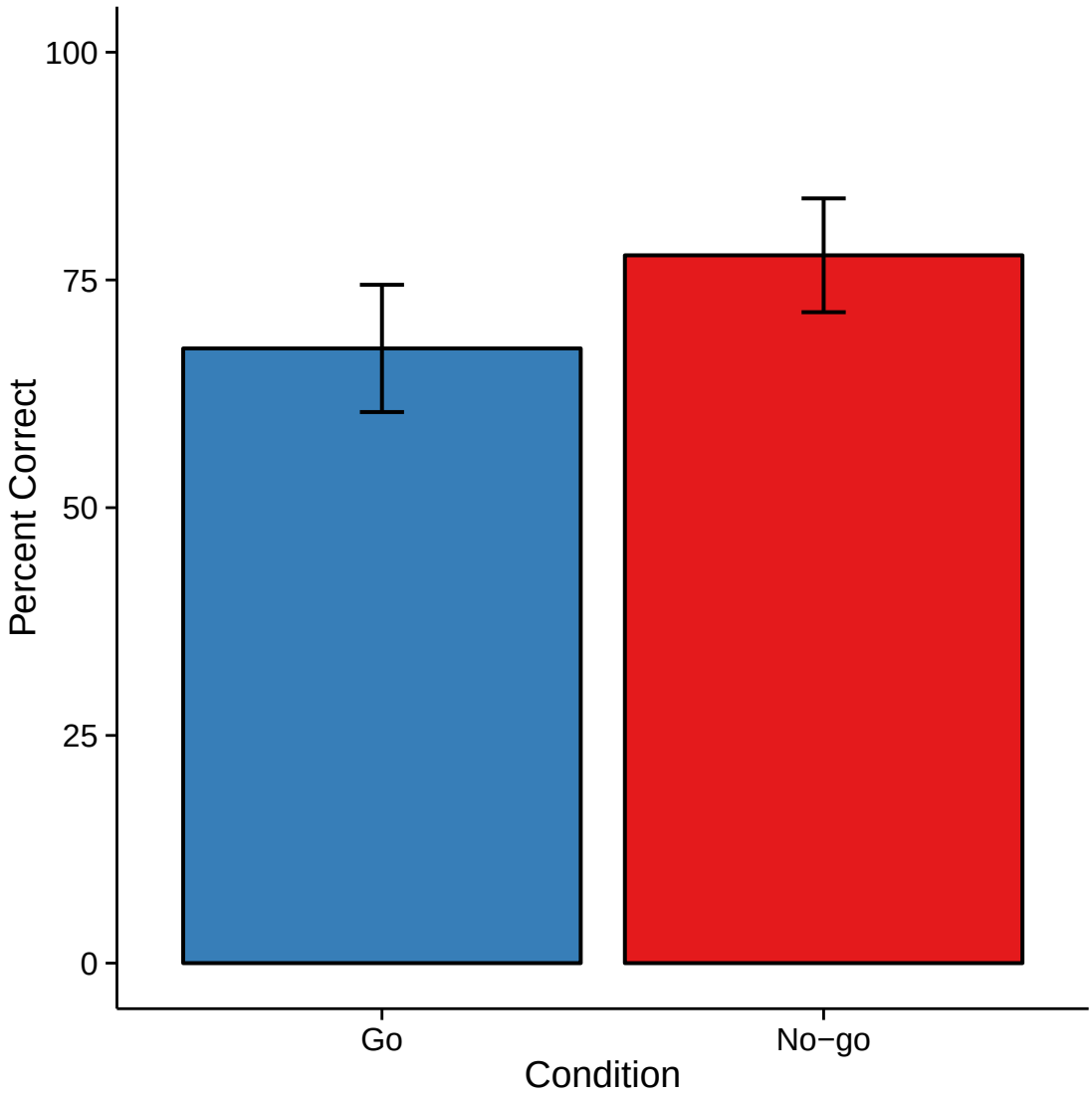

FIGURE 3.5: Bar graph of behavioral performance (percent correct) in Fish/Sharks electrophysiological task. Error bars represent 95% confidence intervals.

similar or different when including incorrect trials. Because of the child-friendly tasks, more than two-thirds of EEG assessments (67%) yielded usable data, in line with prior studies of this age (Bell & Cuevas, 2012). Reasons for EEG missingness included: did not wear cap (9%), refused to play (8%), too many bad trials (i.e., fewer than 9 good trials in any condition; 12%), too many bad channels (i.e., fewer than 100 good channels; 1%), and other technical problem (3%).

A topo plot depicting the ERP waveforms grand-averaged across participants at each electrode site for the go and no-go conditions of the Fish/Sharks ERP task is in Figure 3.6. Grand-averaged ERP waveforms averaged across frontocentral electrodes for the go and no-go conditions are in Figure 3.7. A spatial PCA identified 11 spatial components (i.e., independent electrode clusters) accounting for 90% of the variance in the ERP waveform. A separate temporal PCA identified 8 temporal components (i.e., independent time windows) accounting for 96% of the variance. A spatial PCA on the ERP data reduced by the temporal PCA (i.e., a temporo-spatial PCA) identified 7 spatial components accounting for 84% of the variance. The peak latency of the temporo-spatial component corresponding to the N2 was 392 ms (see Figure 3.8). The electrode cluster for the frontocentral spatial component corresponding to the N2 is displayed in Figure 3.9. The peak latency of the frontocentral spatial component corresponding to the N2 was 390 ms (see Figure 3.10). To identify each individual's peak latency of the N2 component, we identified each individual's latency to their peak minimum amplitude (because the N2 is a negative-going component) during the range of 390 ± 50 ms, i.e., 340–440 ms.

Descriptive statistics of children's N2 amplitudes and latencies are in Table 3.1. Pearson

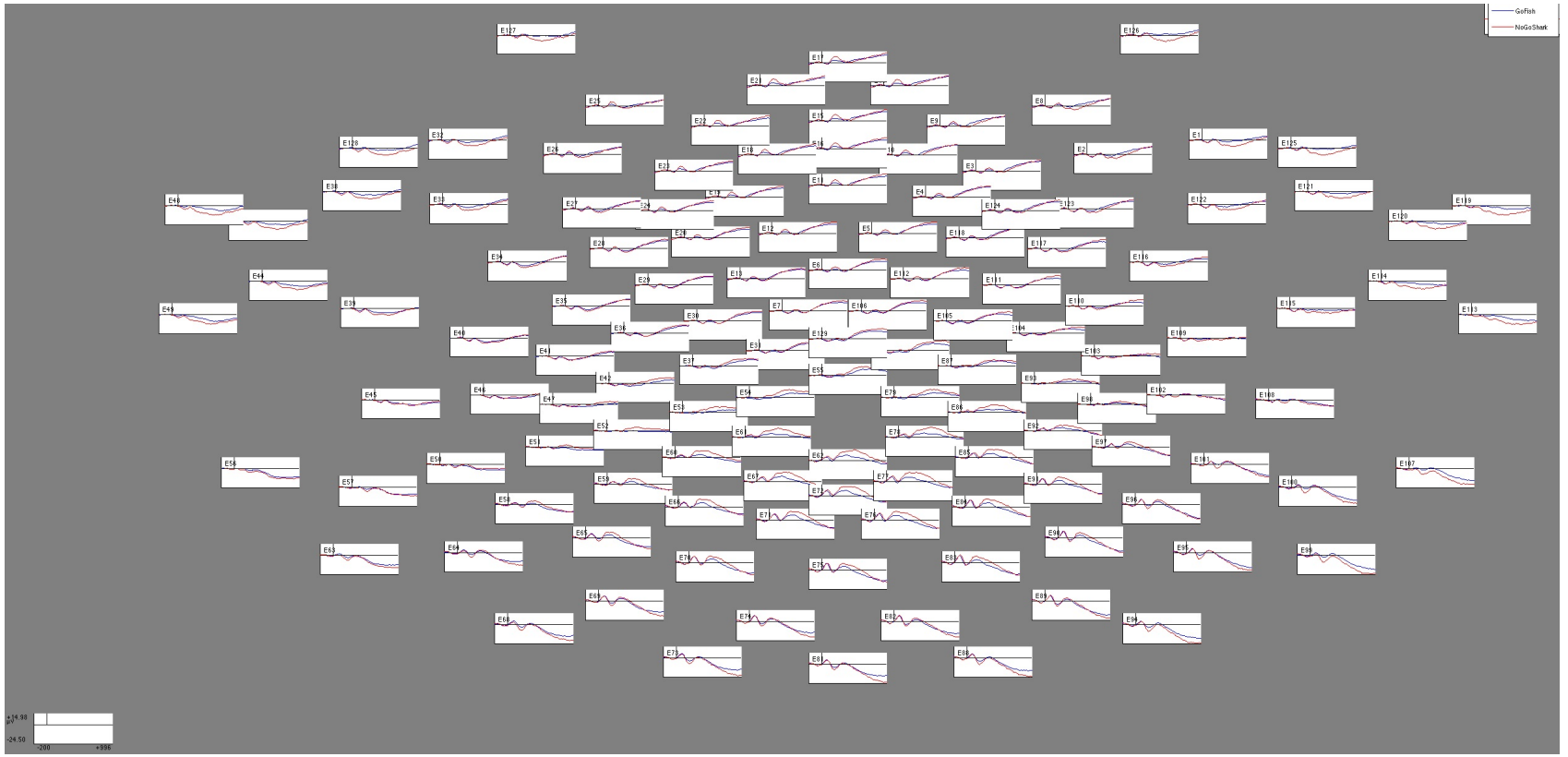

FIGURE 3.6: Topo plot of grand-averaged ERP waveforms for the go (blue) and no-go (red) conditions of the Fish/Sharks ERP task. The negative-going wave corresponding to the N2 can be observed in the frontocentral electrodes. Frontal electrodes (e.g., E17) are located at top of diagram, posterior electrodes (e.g., E81) at bottom.

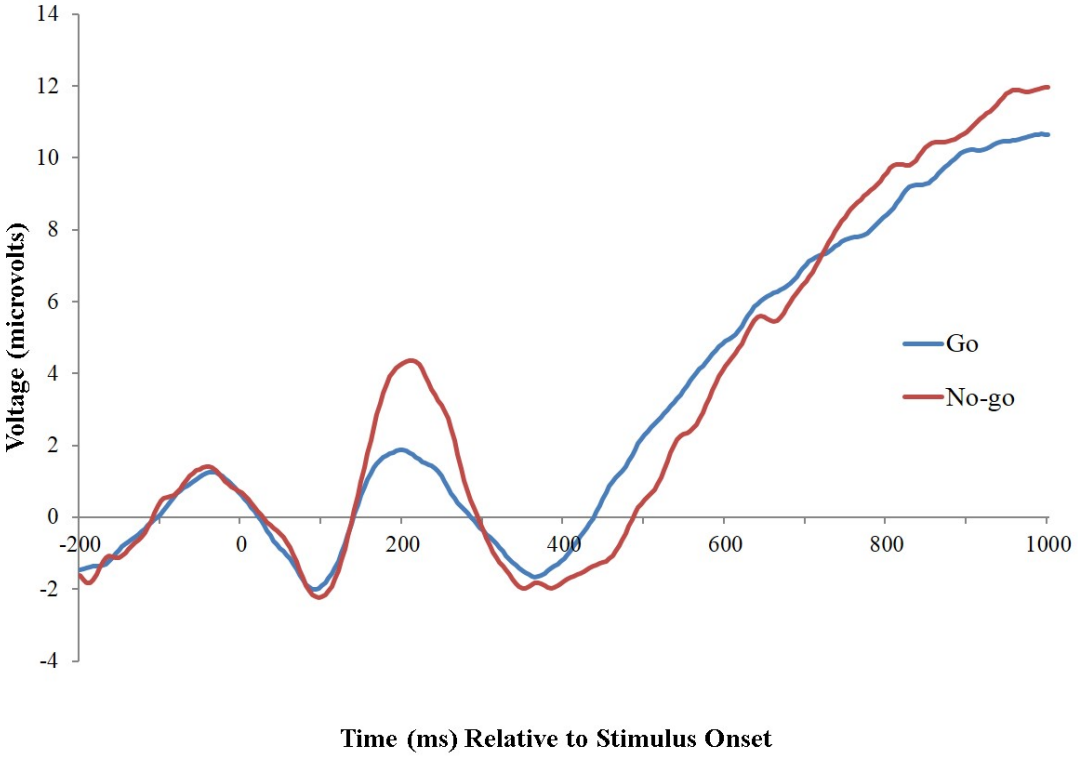

FIGURE 3.7: Grand-averaged ERP waveforms averaged across frontocentral electrodes for the go and no-go conditions of the Fish/Sharks ERP task. Waveforms were averaged across electrodes from the frontocentral electrode cluster identified by the spatial PCA (see Figure 2.8).

correlations of children's N2 amplitudes and latencies are in Table 3.2. N2 amplitudes/latencies were not significantly associated with P3a amplitudes/latencies. Although no-go N2 amplitudes did not appear to be larger than go N2 amplitudes in the grand-averaged waveform, the grand-averaged waveform is composed of multiple underlying components that can influence the morphology of each other. An earlier P1 positivity in the waveform appeared to be more positive in the no-go than go condition and may have deflected the no-go N2 upward. The PCA seeks to separate the independent contributions of these

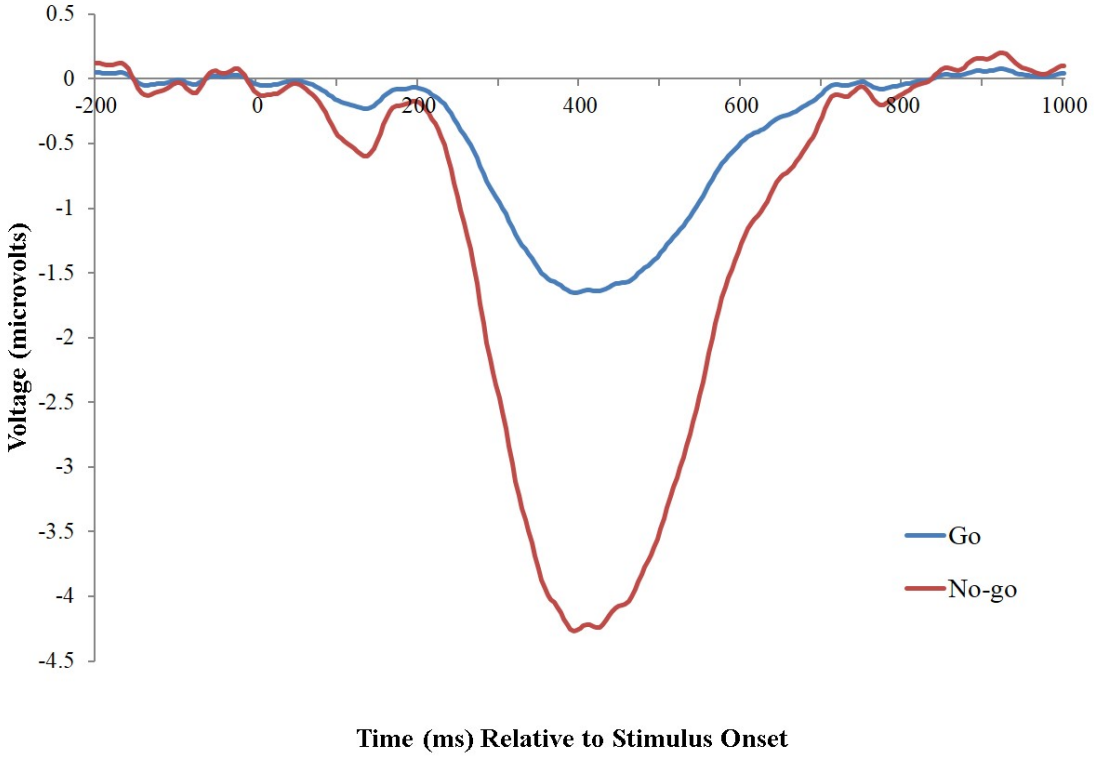

FIGURE 3.8: N2 ERP component in Fish/Sharks Task isolated by temporospatial PCA. Waveforms were averaged across electrodes from the frontocentral electrode cluster identified by the spatial PCA (see Figure 3.9).

underlying components, which allows us to estimate individuals' N2 amplitudes more accurately. When examining the N2 PCA component, children's no-go N2 amplitudes tended to be larger (i.e., more negative) than their go N2 amplitudes ($t[52] = -2.31$, $p = .025$), providing some evidence that the no-go N2 may be related to inhibitory processing. We did not observe strong cross-time continuity in N2 amplitudes or latencies. Among the 13 children with usable ERP data on the Fish/Sharks task at multiple assessments (out of 20 children with multiple EEG assessments), the cross-time continuity of the N2 amplitude was $r(11) = .41$ ($p = .167$) and N2 latency was $r(8) = -.04$ ($p = .908$). Correlations between the N2 and child's age were $r(49) = .03$ ($p = .832$) for N2 amplitudes and $r(45) = -.07$

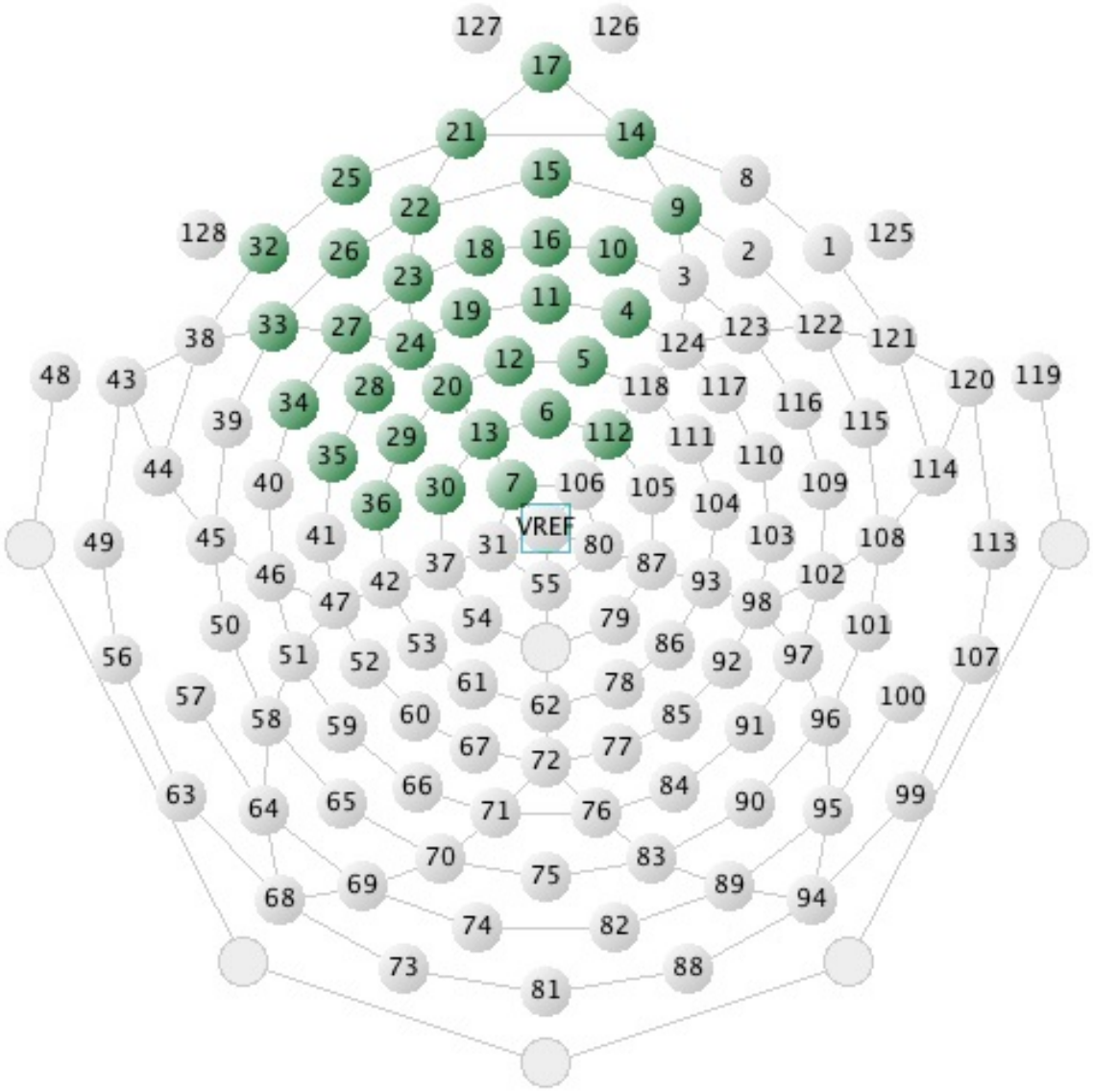

FIGURE 3.9: Electrode cluster for the frontocentral spatial component in the Fish/Sharks ERP task corresponding to the N2. Electrodes in green represent those electrodes with loadings of .40 or greater on the spatial PCA component.

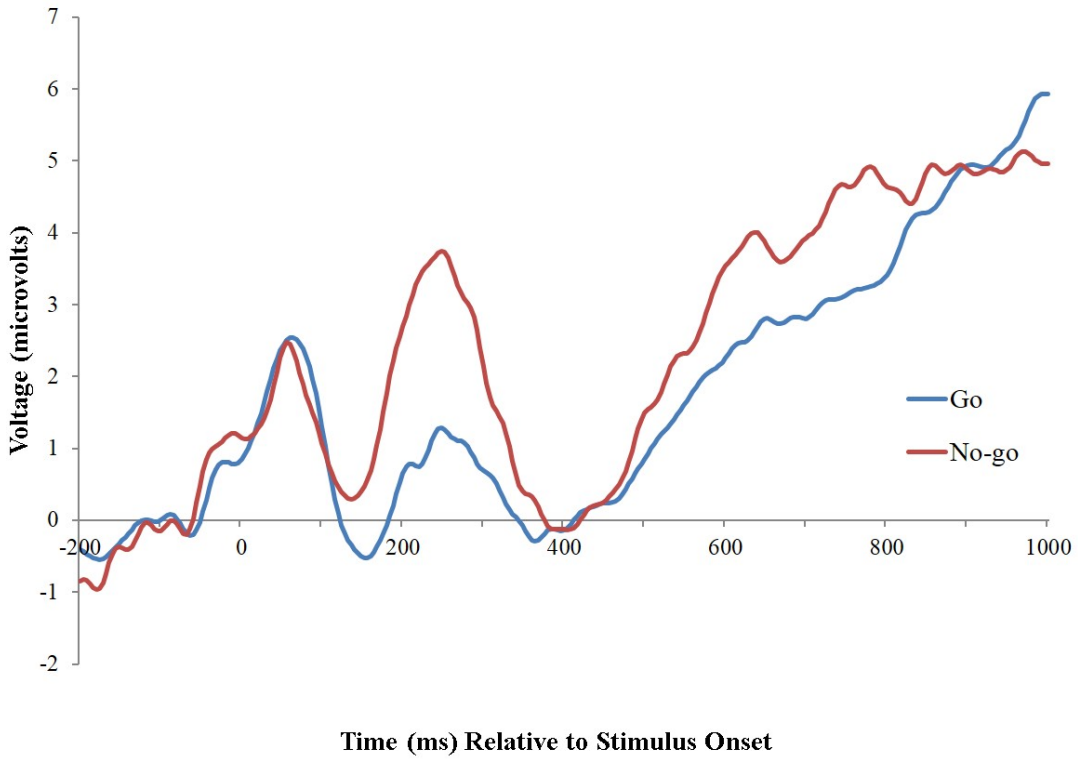

FIGURE 3.10: Frontocentral ERP waveform in Fish/Sharks task isolated by spatial PCA. Waveforms were averaged across electrodes from the frontocentral electrode cluster identified by the spatial PCA (see Figure 3.9).

($p = .663$) for N2 latencies, suggesting that there were no significant developmental changes in N2 amplitudes or latencies in the current sample.[1]

A topo plot depicting the ERP waveforms grand-averaged across participants at each electrode site for the target and frequent conditions of the auditory oddball ERP task are in Figure 3.11. Grand-averaged ERP waveforms averaged across posterior electrodes for the target and frequent conditions are in Figure 3.12. A spatial PCA identified 13 spatial components accounting for 87% of the variance in the ERP waveform. A separate temporal PCA identified 13 temporal components accounting for 95% of the variance. A spatial PCA on the ERP data reduced by the temporal PCA (i.e., a temporo-spatial PCA) identified 7 spatial components accounting for 84% of the variance. The peak latency of the temporo-spatial component corresponding to the P3a was 436 ms (see Figure 3.13). The electrode cluster for the posterior spatial component corresponding to the P3a is displayed in Figure 3.14. The peak latency of the posterior spatial component corresponding to the P3a was 442 ms (see Figure 3.15). To identify each individual's peak latency of the P3a component, we identified each individual's latency to their peak maximum amplitude (because the P3a is a positive-going component) during the range of 442 ± 50 ms, i.e., 392–492 ms.

Descriptives of children's P3a amplitudes and latencies are in Table 3.1. Pearson correlations of children's P3a amplitudes and latencies are in Table 3.2. Unexpectedly, children's target P3a amplitudes were not significantly larger than their frequent P3a amplitudes ($t[83] = 0.71$, $p = .481$), although the effect was in the direction hypothesized. We did not observe strong cross-time continuity in P3a amplitudes or latencies. Among the 20 children

[1]Latencies were unable to be estimated with the ERP PCA Toolkit for some children with values for amplitudes.

TABLE 3.1: Study 2: Descriptive Statistics of Children's ERP Components.

	P3a					N2				
	Tgt Amp	Frq Amp	Amp Diff	Tgt Lat	Frq Lat	Go Amp	No-Go Amp	Amp Diff	Go Lat	No-Go Lat
N	84.00	84.00	84.00	83.00	79.00	53.00	53.00	53.00	51.00	49.00
M	6.76	6.17	0.59	442.70	439.95	−3.25	−7.18	−3.94	386.75	395.43
SD	6.70	6.74	7.61	24.95	24.33	10.19	9.90	12.43	28.02	24.17
min	−9.48	−16.00	−28.39	400.00	400.00	−25.41	−30.26	−60.26	348.00	352.00
max	19.92	25.09	16.53	488.00	484.00	46.93	20.28	22.80	436.00	436.00

Note. "Amp" = amplitude, "Lat" = latencies, "Diff" = difference, "Tgt" = target, "Frq" = frequent. Amplitudes are in microvolts, latencies are in milliseconds. P3a amplitude difference reflects target P3a amplitude − frequent P3a amplitude. N2 amplitude difference reflects no-go N2 amplitude − go N2 amplitude.

TABLE 3.2: Study 2: Pearson Correlations of Children's ERP Components.

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.
1. Target P3a amplitude	–									
2. Frequent P3a amplitude	.36**	–								
3. P3a amplitude difference	.56***	−.57***	–							
4. Target P3a latency	−.11	.11	−.19†	–						
5. Frequent P3a latency	−.02	−.12	.10	−.05	–					
6. Go N2 amplitude	.01	.03	−.02	−.34*	−.10	–				
7. Nogo N2 amplitude	−.05	.24†	−.26†	.14	−.34*	.23†	–			
8. N2 amplitude difference	−.05	.17	−.19	.39**	−.19	−.63***	.60***	–		
9. Go N2 latency	−.14	−.18	.03	.04	−.14	−.17	−.08	.07	–	
10. Nogo N2 latency	−.10	−.08	−.02	−.12	−.09	.02	−.17	−.16	.03	–

Note. $^{\dagger}p < .10$. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$. Correlations are two-tailed.

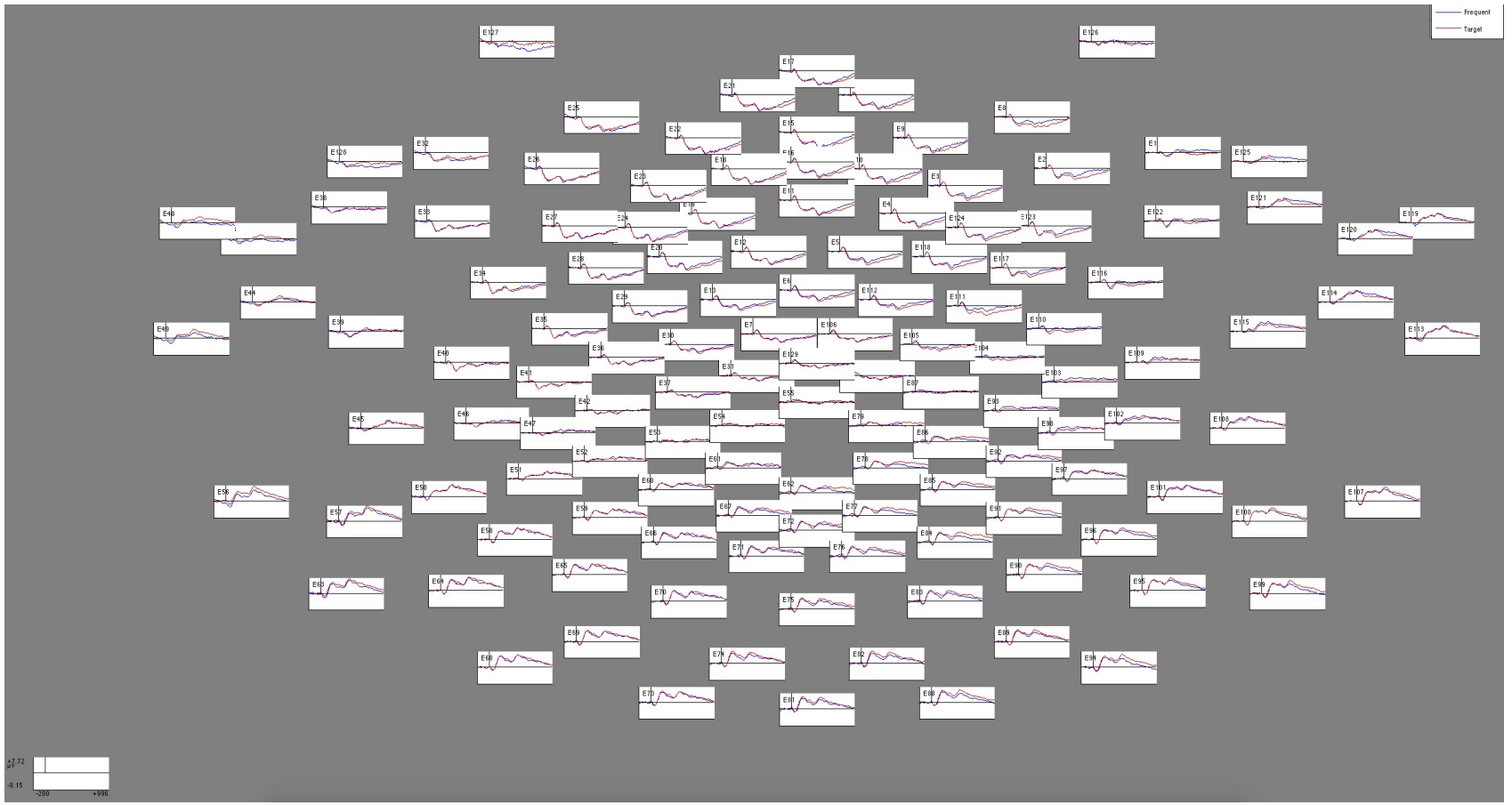

FIGURE 3.11: Topo plot of grand-averaged ERP waveforms for the target (red) and frequent (blue) conditions of the auditory oddball task. The positive-going wave corresponding to the P3a can be observed in the posterior electrodes. Frontal electrodes (e.g., E17) are located at top of diagram, posterior electrodes (e.g., E81) at bottom.

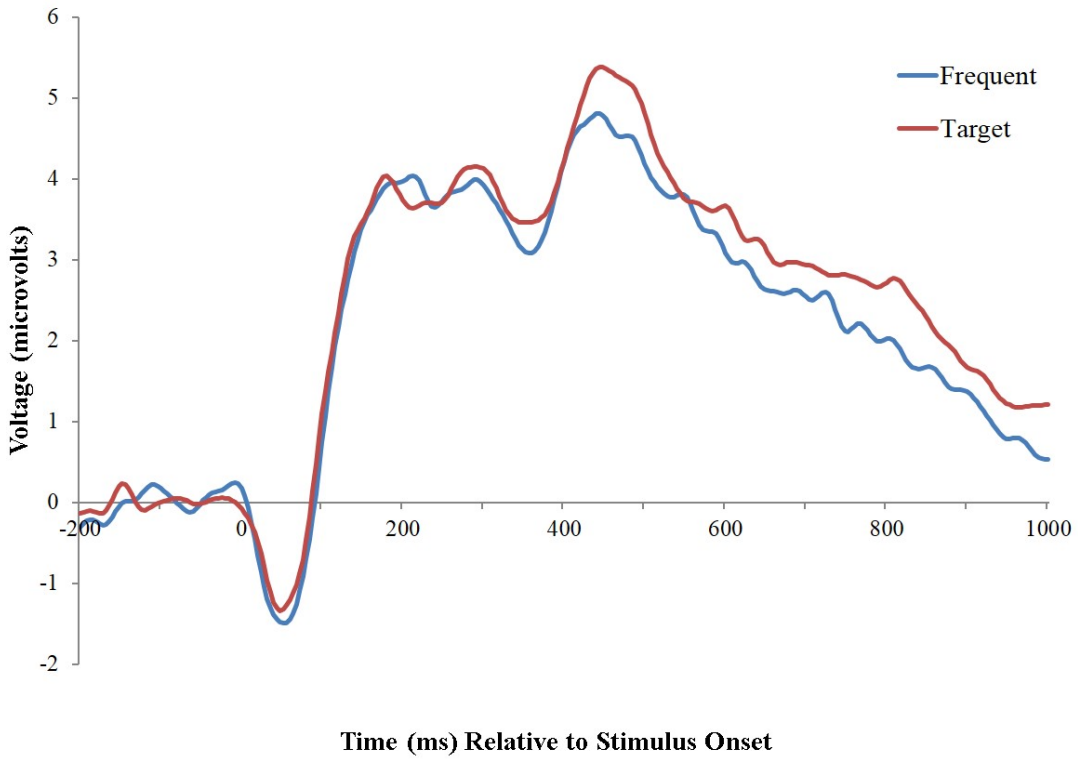

FIGURE 3.12: Grand-averaged ERP waveforms averaged across posterior electrodes for the target and frequent conditions of the oddball task. Waveforms were averaged across electrodes from the posterior electrode cluster identified by the spatial PCA (see Figure 3.14).

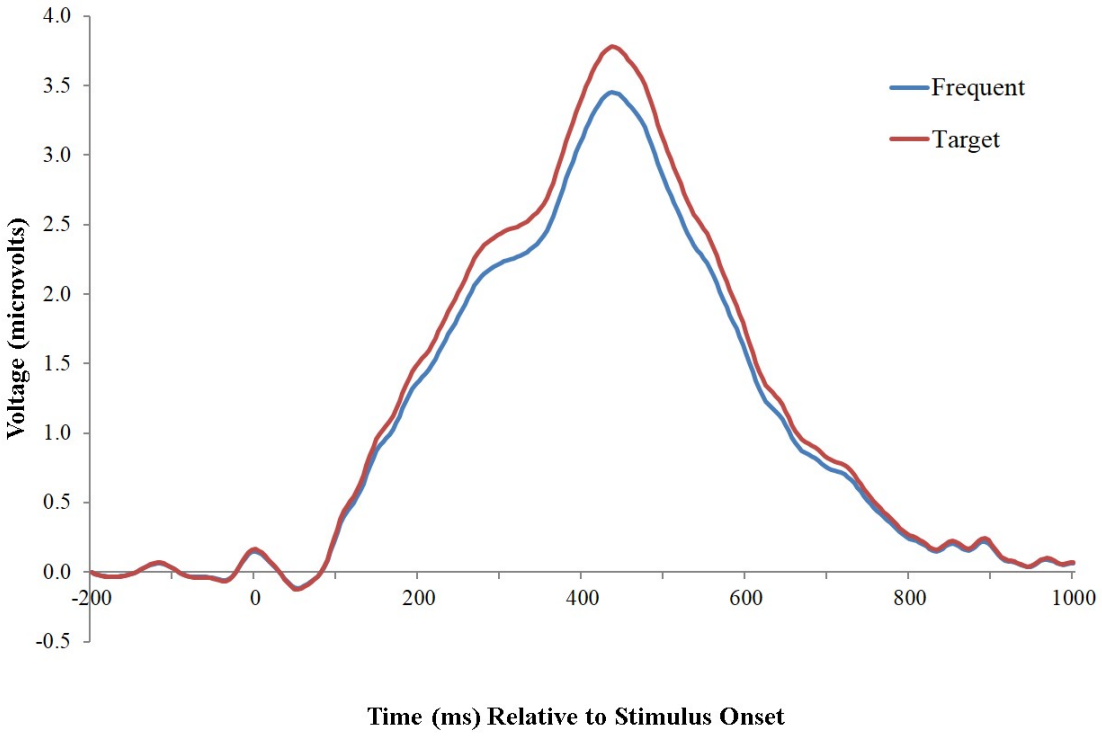

FIGURE 3.13: P3b ERP component in oddball task isolated by temporospatial PCA. Waveforms were averaged across electrodes from the posterior electrode cluster identified by the spatial PCA (see Figure 3.14).

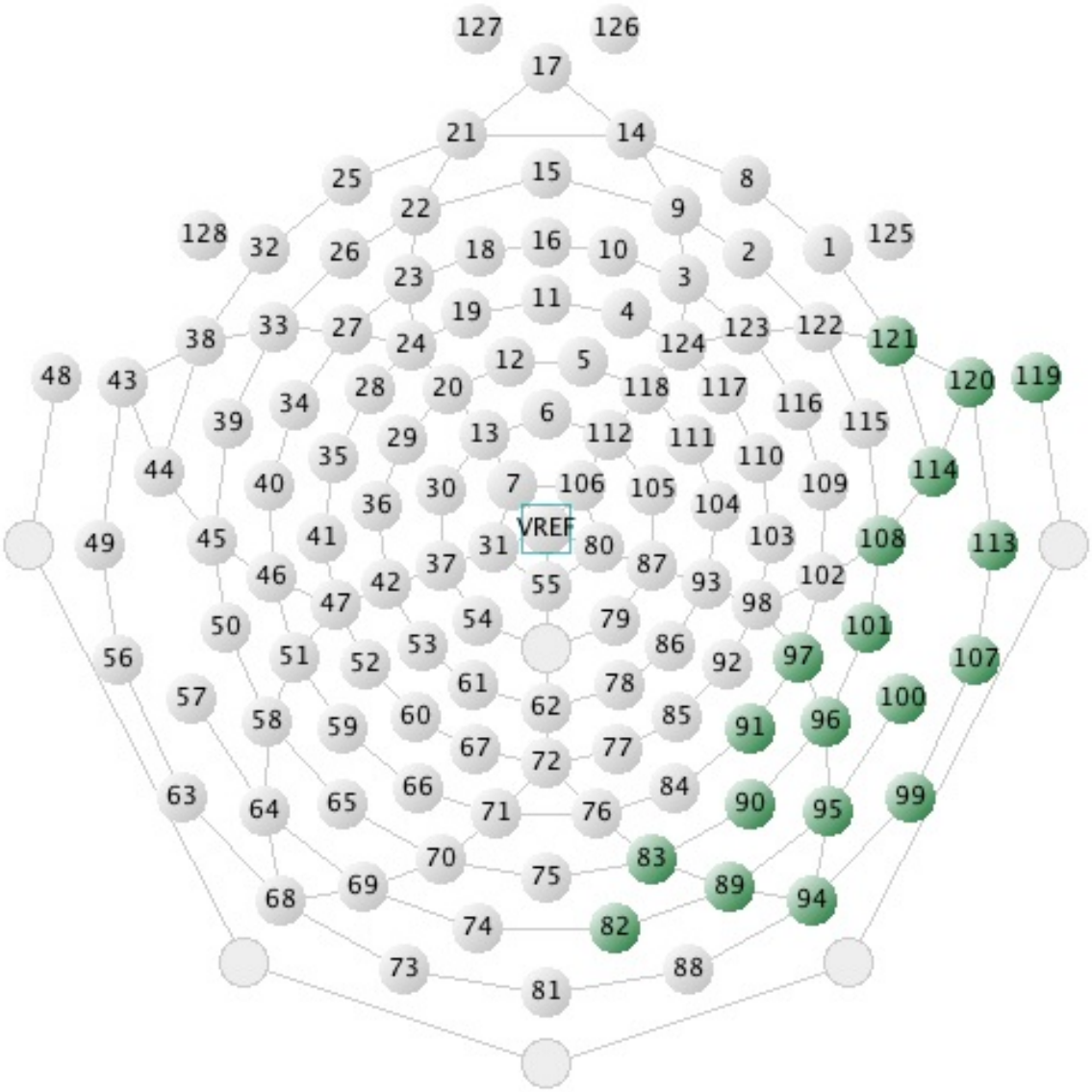

FIGURE 3.14: Electrode cluster for the posterior spatial component in the oddball task corresponding to the P3a. Electrodes in green represent those electrodes with loadings of .40 or greater on the spatial PCA component.

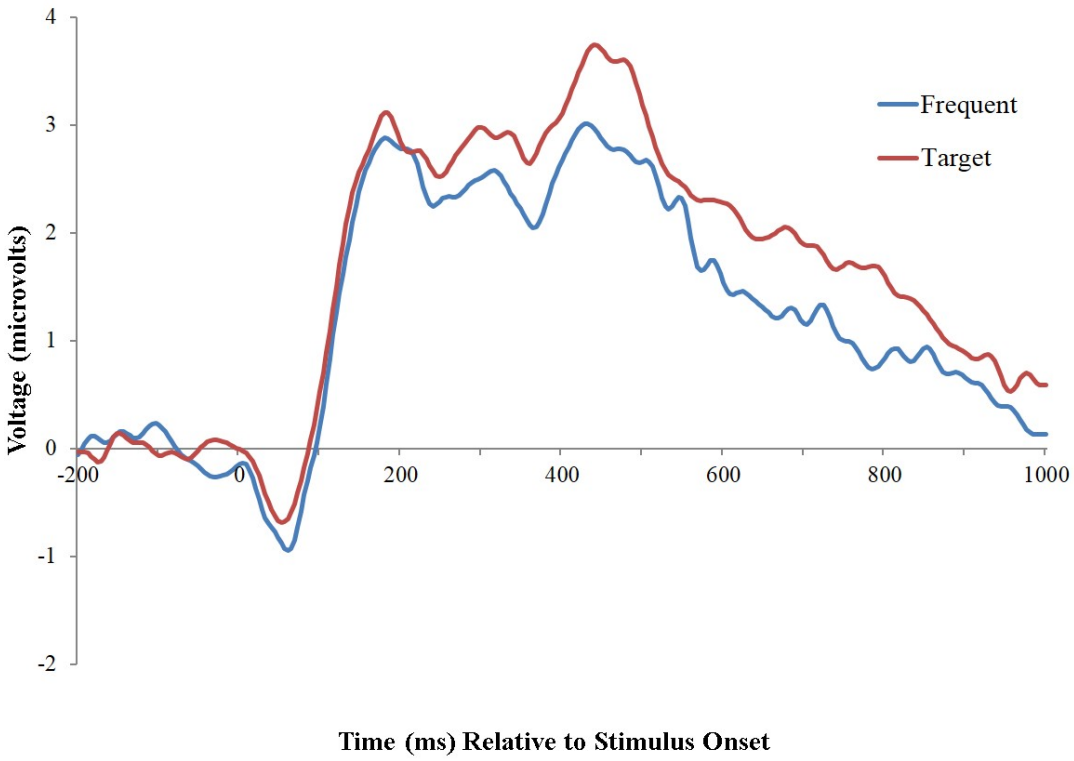

FIGURE 3.15: Posterior ERP waveform in oddball task isolated by spatial PCA. Waveforms were averaged across electrodes from the posterior electrode cluster identified by the spatial PCA (see Figure 3.14).

with usable ERP data on the oddball task at multiple assessments (out of 20 children with multiple EEG assessments), the cross-time continuity of the P3a amplitude was $r(18) = .13$ ($p = .584$) and P3a latency was $r(17) = .14$ ($p = .559$). Correlations between the P3a and child's age were $r(80) = .00$ ($p = .977$) for P3a amplitudes and $r(79) = .02$ ($p = .841$) for P3a latencies, suggesting that there were no significant developmental changes in P3a amplitudes or latencies in the current sample.

3.1.2.1.4 EEG Data Processing. The processing of EEG data was similar to the data processing in Study 1 (see Section 2.1.2.1.4). For calculating frontal alpha power, we selected the frontal electrode cluster based on a spatial PCA, which resulted in the frontocentral spatial component corresponding to the N2 (see Figure 3.9). Log-transformed power values across frequencies at frontal electrodes in each task are depicted in Figure 3.16. The power spectrum decomposition showed similar power (amplitude in log-transformed squared microvolts) across the oddball and Fish/Sharks tasks, with higher power at low than at high frequencies. Descriptives of children's frontal power and frontal asymmetry scores are in Table 3.3. Pearson correlations of children's frontal power and frontal asymmetry scores are in Table 3.4. Frontal alpha power was strongly positively associated across the Fish/Sharks and oddball tasks, and frontal asymmetry was moderately positively associated across the tasks.

We observed limited evidence of cross-time continuity of EEG measures in our sample. Among the 20 children with multiple EEG assessments, the cross-time continuity of frontal alpha power was $r(18) = .37$ ($p = .113$) in the oddball task and $r(11) = .63$ ($p = .021$) in the Fish/Sharks task. The cross-time continuity of frontal asymmetry was $r(18) = -.39$

(p = .091) in the oddball task and $r(11) = -.05$ (p = .873) in the Fish/Sharks task. Consistent with Study 1 and a previous study (Vuga et al., 2008), we observed greater cross-time continuity of frontal alpha power than frontal asymmetry. Correlations between frontal alpha power and child's age were $r(80) = -.07$ (p = .530) in the oddball task and $r(49) = -.42$ (p = .002) in the Fish/Sharks task. Correlations between left frontal asymmetry and child's age were $r(80) = -.07$ (p = .518) in the oddball task and $r(49) =$ .06 (p = .659) in the Fish/Sharks task. Frontal alpha power decreased with age in the Fish/Sharks task; otherwise, there were no significant developmental changes in the EEG measures examined.

3.1.2.1.5 Time-Frequency Data Processing. The processing of time-frequency data was similar to the data processing in Study 1 (see Section 2.1.2.1.5). Because we were particularly interested in the theta activity linked to the no-go N2 and oddball target P3 ERPs, we examined the EEG data in the no-go and oddball target conditions only. For time-frequency analysis, we examined frontal theta activity (4–5 Hz) from the decomposition of frequencies at the time frames corresponding to the no-go N2 (340–440 ms) and oddball P3a (392–492 ms). Time-frequency analysis plots are in Figures 3.17 and 3.18 for the Fish/Sharks and oddball tasks, respectively.

Descriptive statistics of children's time-frequency components are in Table 3.3. Pearson correlations of children's time-frequency components are in Table 3.4. N2-related frontal theta activity was not significantly associated across the Fish/Sharks and oddball tasks. Among the 20 children with multiple EEG assessments, there was no evidence of cross-time continuity. The cross-time continuity of P3a-related frontal theta activity in the oddball task

Table 3.3: Study 2: Descriptive Statistics of Children's EEG and Time-Frequency Components.

	Oddball			Fish/Sharks		
	Frontal Power	Frontal Asymmetry	Frontal TF	Frontal Power	Frontal Asymmetry	Frontal TF
N	84.00	84.00	84.00	53.00	53.00	53.00
M	3.67	−0.09	1.04	4.28	−0.07	0.73
SD	0.59	0.41	4.69	0.59	0.36	4.91
min	1.90	−1.00	−10.45	3.06	−0.85	−7.08
max	5.21	0.67	13.08	5.69	0.64	14.84

Note. "TF" = time-frequency activity corresponding to timing of P3a (oddball) or N2 (Fish/Sharks), with values in decibels. Power values were log-transformed. Frontal power and asymmetry in alpha frequency range. Frontal time-frequency in theta frequency range. Frontal alpha asymmetry reflects right frontal alpha power − left frontal alpha power.

Table 3.4: Study 2: Pearson Correlations of Children's EEG and Time-Frequency Components.

	1.	2.	3.	4.	5.	6.
1. Oddball Frontal Alpha Power	–					
2. Oddball Frontal Alpha Asymmetry	−.10	–				
3. Oddball P3a-Related Frontal Theta	.21†	.04	–			
4. Fish/Sharks Frontal Alpha Power	.54***	.18	.18	–		
5. Fish/Sharks Frontal Alpha Asymmetry	.10	.25†	.16	.04	–	
6. Fish/Sharks N2-Related Frontal Theta	−.02	−.03	−.04	.07	.05	–

Note. $^{\dagger}p < .10$. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$. Correlations are two-tailed.

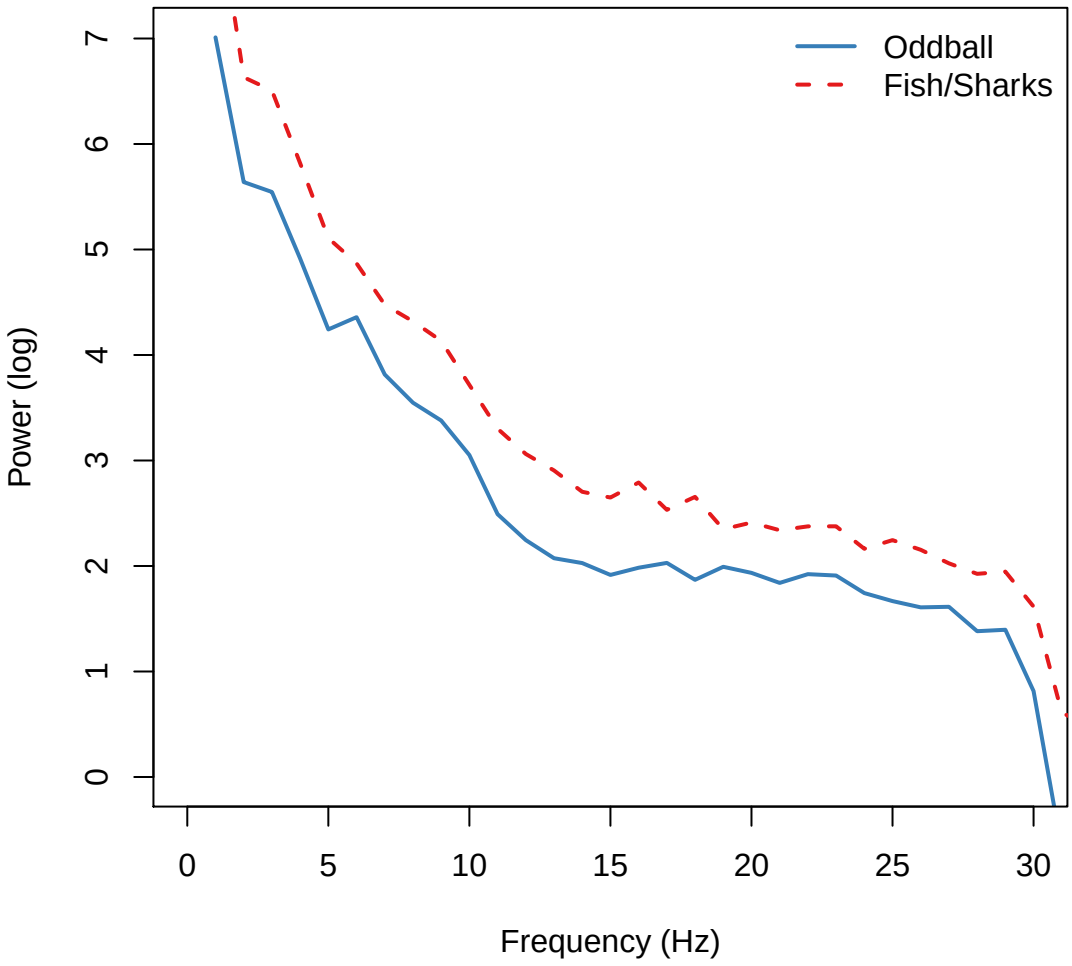

FIGURE 3.16: Power spectrum decomposition of the EEG waveforms. Log-transformed power values across frequencies at frontal electrodes (see Figure 3.9) in the Fish/Sharks and oddball tasks.

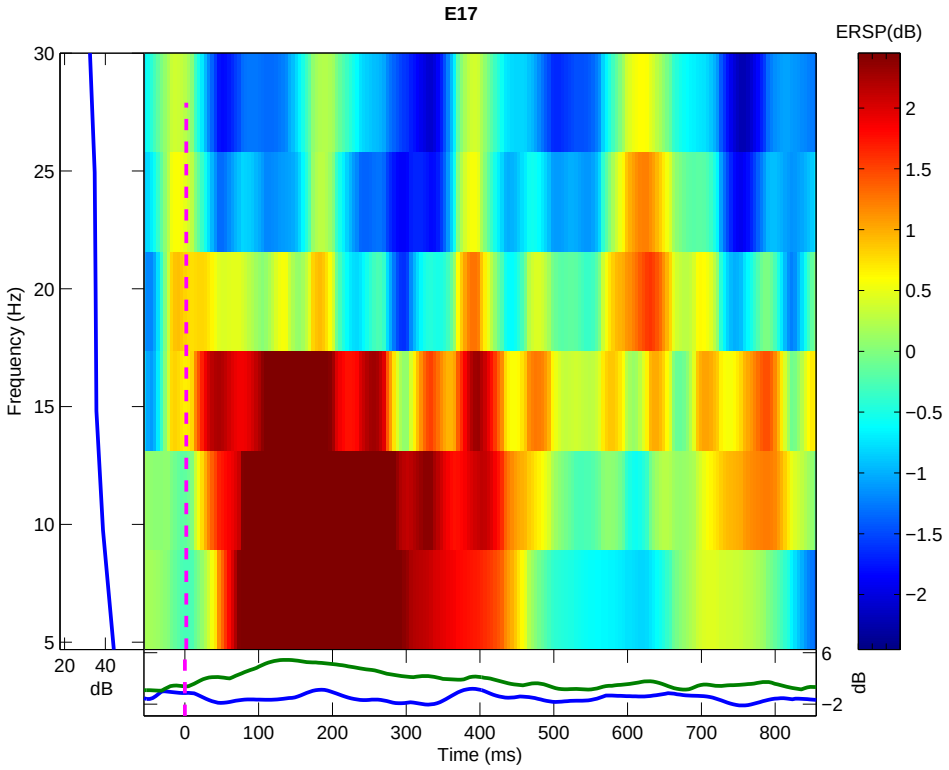

FIGURE 3.17: Time-frequency analysis plot of event-related spectral perturbation values (in decibels) from a frontal electrode (E17) in the Fish/Sharks task. Actual time-frequency estimates were averaged across electrodes from the frontal electrode cluster identified by the spatial PCA (see Figure 3.9). Frontal N2-related theta activity corresponds to high power from 340 to 440 ms in the 4–5 Hz frequency band.

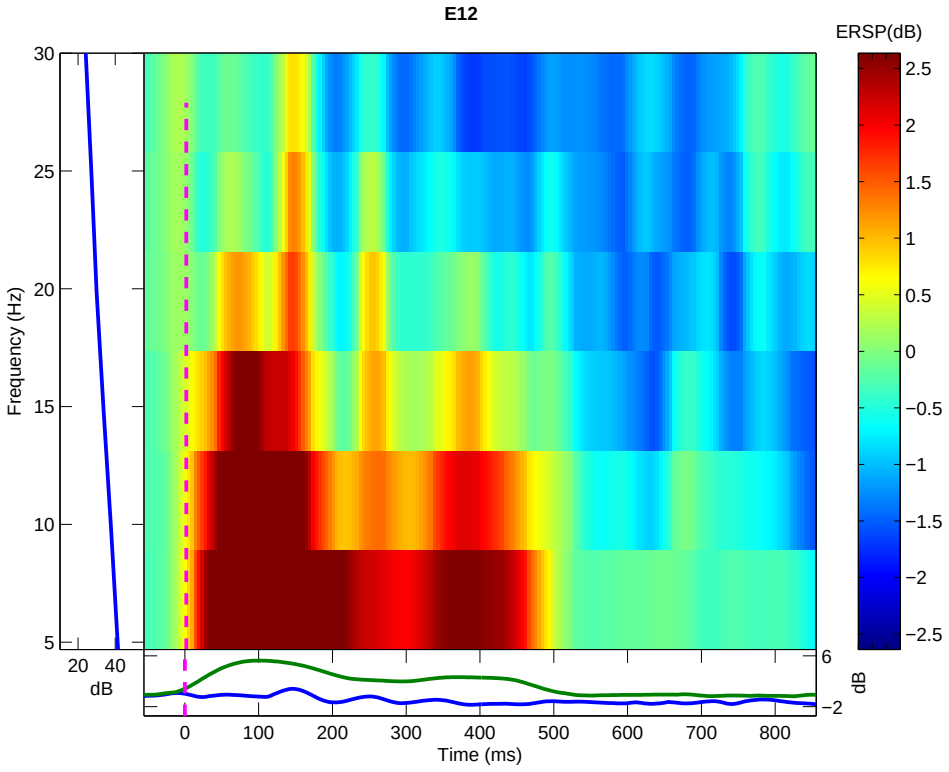

FIGURE 3.18: Time-frequency analysis plot of event-related spectral perturbation values (in decibels) from a frontal electrode (E12) in the oddball task. Actual time-frequency estimates were averaged across electrodes from the frontal electrode cluster identified by the spatial PCA (see Figure 3.9). Frontal P3a-related theta activity corresponds to high power (red shading) from 392 to 492 ms in the 4–5 Hz frequency band.

was $r(18) = .06$ ($p = .807$). The cross-time continuity of N2-related frontal theta frontal activity in the Fish/Sharks task was $r(11) = -.51$ ($p = .072$). The correlation between P3a-related frontal theta activity and child's age in the oddball task was $r(80) = -.02$ ($p = .866$). The correlation between N2-related frontal theta activity and child's age in the Fish/Sharks task was $r(49) = .06$ ($p = .653$), suggesting that there were no significant developmental changes in these components in the current sample.

3.1.2.2 Self-Regulation

The tasks measuring self-regulation were similar to those in Study 1 (see Section 2.1.2.2).

3.1.2.3 Externalizing Behavior Problems

The measures of externalizing behavior problems were similar to those in Study 1 (see Section 2.1.2.3).

3.1.2.4 Missingness

We examined whether children's missingness differed systematically as a function of other variables, including temperament (as reported by parents on the CBQ), externalizing problems, sex, and SES. Compared to children who provided usable electrophysiological data, children who did not provide usable data did not differ in terms of secondary caregiver-reported externalizing problems ($t[8.43] = 0.89$, $p = .396$), parent-reported externalizing problems ($t[17.82] = 0.13$, $p = .898$), fearful temperament ($t[17.55] = -0.20$, $p = .844$), impulsive temperament ($t[17.47] = 0.80$, $p = .435$), sex ($\chi^2[1] = 0.32$, $p = .792$), or SES

($t[8.24] = -0.56$, $p = .591$). Moreover, several children ($n = 10$) who provided usable data had scores above the normed 80th percentile on externalizing problems (i.e., T-score > 58). As in Study 1, however, younger children were more likely to be missing electrophysiological data than older children ($t[28.33] = -2.72$, $p = .011$). As a result, we included the child's age as a covariate in the clustered regression models. With the exception of missingness by the child's age, however, there was no evidence of any systematic missingness in variables of interest that would compromise the representativeness of the sample and generalizability of findings.

3.1.3 Procedure

The procedures were similar to those in Study 1 (see Section 2.1.3), except with two different EEG tasks: In Study 2, we used the P3a oddball (that did not require a behavioral response) and Fish/Sharks tasks unlike in Study 1, in which we used the P3b oddball (that involved a behavioral response to the target stimulus) and Bird/Alligator tasks.

3.1.4 Statistical Analysis

3.1.4.1 Statistical Models

Similar statistical analyses were planned to Study 1 (see Section 2.1.5.1). In the concurrent regression models, we included covariates for sex, age, the number of bad channels, the number of trials kept in the condition of interest (no-go or target), and behavioral percent correct in the no-go condition (when examining neurophysiological variables from the Fish/Sharks task). In the lagged regression models, in addition to these covariates,

we added autoregressive controls of the dependent variable in order to predict rank-order change in self-regulation and externalizing problems over time. We also examined receiver operating characteristic (ROC) curves to determine the sensitivity and specificity of neurophysiological measures in predicting externalizing problems. To examine the tradeoff between sensitivity and specificity in the context of an ROC curve, the outcome must be binary. We dichotomized externalizing problems by comparing high levels of externalizing problems (above the normed 80th percentile on externalizing problems, i.e., T-score > 58) to lower levels of externalizing problems. Because of the greater rate of longitudinal follow-up in Study 2 compared to Study 1, we used longitudinal mediation models (i.e., cross-lagged panel models) as proposed by Cole and Maxwell (2003) to clarify the developmental process. Longitudinal panel models are important for testing mediation and causal inference (Little, Preacher, Selig, & Card, 2007).

3.1.4.2 Power

There is an increase in power to detect effects in repeated measures designs (B. O. Muthén & Curran, 1997). ERPs tend to have less measurement error and higher reliability than behavioral measures (Räikkönen et al., 2003), resulting in larger effect sizes that permit smaller sample sizes. Sixteen prior studies examining the association between the N2 ERP and self-regulation or externalizing problems had a medium effect size ($d = 0.46$) on average (see Table 1.1). With usable data on 64 children, we would have low power (.44) to detect a simple bivariate association of this magnitude at a given age ($\alpha = .05$, two tailed). However, with longitudinal data, we would have higher power (.78) to detect an association of this magnitude (Scherbaum & Ferreter, 2009). Fewer secondary caregivers than parents

reported on children's behavior problems because some families did not have a secondary caregiver that spent regular time with the child, and some caregivers did not complete the measure. As a result, power is likely low to detect associations between neurophysiology and secondary caregiver-reported behavior problems.

3.2 Results

3.2.1 Association Between Neurophysiology and Self-Regulation

3.2.1.0.1 ERPs and Self-Regulation. Pearson correlations of children's ERP components with their concurrent self-regulation are in Table 3.5. There were no concurrent linear associations between ERPs and self-regulation that were consistent with hypotheses. Several concurrent associations between ERPs and self-regulation were inconsistent with hypotheses. Smaller (less negative) N2 amplitudes and smaller N2 difference scores were associated with better performance on Bird/Alligator, and to a trend-level, with better performance on Fish/Sharks. Longer N2 latencies were marginally significantly associated with better performance on Shape Stroop.

The apparent lack of concurrent correlations between N2 amplitudes and self-regulation may have been masked by a nonlinear association. N2 amplitudes showed marginally stronger quadratic than linear fit in associations with performance on Shape Stroop ($F[1] = 3.22$, $p = .079$) and Token Sort ($F[1] = 3.69$, $p = .061$). Quadratic associations of N2 amplitudes with Shape Stroop ($p = .909$) and Token Sort ($p = .255$) did not remain significant when examining Spearman's rho, suggesting that the quadratic association may have owed, in part, to outliers. The clustered linear and quadratic regression results of the association

TABLE 3.5: Study 2: Pearson Correlations of Children's ERP Components with their Self-Regulation.

	P3a					N2				
	Tgt Amp	Frq Amp	Amp Diff	Tgt Lat	Frq Lat	Go Amp	No-Go Amp	Amp Diff	Go Lat	No-Go Lat
Bird/Alligator	.00	.11	−.10	.19	.15	−.13	.31*	.35*	.05	−.22
Shape Stroop	.15	.02	.12	.03	.08	.02	.00	−.02	.11	.29†
Grass/Snow	.08	.01	.06	−.14	.01	−.13	.04	.14	−.15	−.11
Token Sort	.12	−.01	.11	−.05	−.11	−.01	.16	.13	.12	−.23
Sustained Play Attention	−.08	.10	−.15	.06	−.19	−.02	.25	.25	.12	.07
Fish/Sharks	.07	.16	−.08	.15	−.10	−.34*	−.02	.26†	.14	.12

Note. "Amp" = amplitude, "Lat" = latencies, "Diff" = difference, "Tgt" = target, "Frq" = frequent. Amplitudes are in microvolts, latencies are in milliseconds. P3a amplitude difference reflects target P3a amplitude − frequent P3a amplitude. N2 amplitude difference reflects no-go N2 amplitude − go N2 amplitude. Correlations are two-tailed.

of target N2 amplitudes with Shape Stroop and Token Sort are in Table 3.6. Examination of the scatterplots (Shape Stroop: Figure 3.19; Token Sort: Figure 3.20) suggests that there was an inverted-U-shaped association between N2 amplitudes and self-regulation. The quadratic association of N2 amplitudes with Shape Stroop and Token Sort remained significant even after accounting for the nesting of longitudinal data and controlling for covariates. Self-regulation performance was highest among children with middle-range (-10 to 0 μV) N2 amplitudes and was lowest among children with larger (< -10 μV) or smaller N2 (> 0 μV) amplitudes.

Pearson correlations of children's ERP components with their later self-regulation are in Table 3.7. There were several associations between ERPs and later self-regulation that were consistent with hypotheses. Shorter target P3a latencies ($r[21] = -.61$, $p = .002$, see Figure 3.21) and, to a trend-degree, larger (more negative) N2 difference scores ($r[13] = -.45$, $p = .091$, see Figure 3.22) were associated with better performance on Token Sort. P3a latencies remained associated with performance on Token Sort when examining Spearman's rho ($p = .014$), whereas N2 amplitude difference scores did not ($p = .109$), suggesting that the association between N2 amplitude difference scores and Token Sort may have owed to outliers. A clustered regression model examining the association of P3a latencies and N2 amplitude difference scores with later performance on Token Sort is in Table 3.8. The N2 amplitude difference score remained associated with Token Sort after accounting for the nesting of longitudinal data and controlling for covariates and prior levels of performance on Token Sort, whereas P3a latencies did not remain associated with later Token Sort.

Shorter no-go N2 latencies were associated with better later performance on the no-go trials of the Fish/Sharks task ($r[11] = -.74$, $p = .004$, see Figure 3.23). No-go N2 latencies

TABLE 3.6: Study 2: Clustered Linear and Quadratic Regression Examining Association of N2 Amplitude with Performance on Shape Stroop and Token Sort.

	Dependent variable:			
	Shape Stroop		Token Sort	
	Linear	Quadratic	Linear	Quadratic
Intercept	0.186 (0.936)	0.126 (0.827)	−6.910 (91.742)	−12.109 (86.175)
No-Go N2 Amplitude (Linear)	−0.007 (0.011)	−0.025** (0.009)	0.684 (0.861)	−1.016 (0.835)
No-Go N2 Amplitude (Quadratic)		−0.001* (0.001)		−0.126*** (0.038)
Sex	0.301* (0.148)	0.253* (0.128)	5.580 (18.844)	1.202 (18.607)
Age	0.414† (0.250)	0.483* (0.222)	47.792* (20.134)	53.916** (17.975)
Number of Bad Channels	−0.051† (0.031)	−0.056* (0.028)	−2.193 (3.584)	−2.729 (3.311)
Number of No-Go Trials Kept	−0.046† (0.027)	−0.063* (0.030)	1.413 (4.319)	−0.046 (4.083)
Behavioral Percent Correct on No-Go Trials	0.012 (0.008)	0.015† (0.009)	−0.628 (0.733)	−0.390 (0.726)
Observations	47	47	44	44
R^2	0.273	0.383	0.194	0.290
Adjusted R^2	0.164	0.272	0.064	0.152

Note. $^{\dagger}p < .10$. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

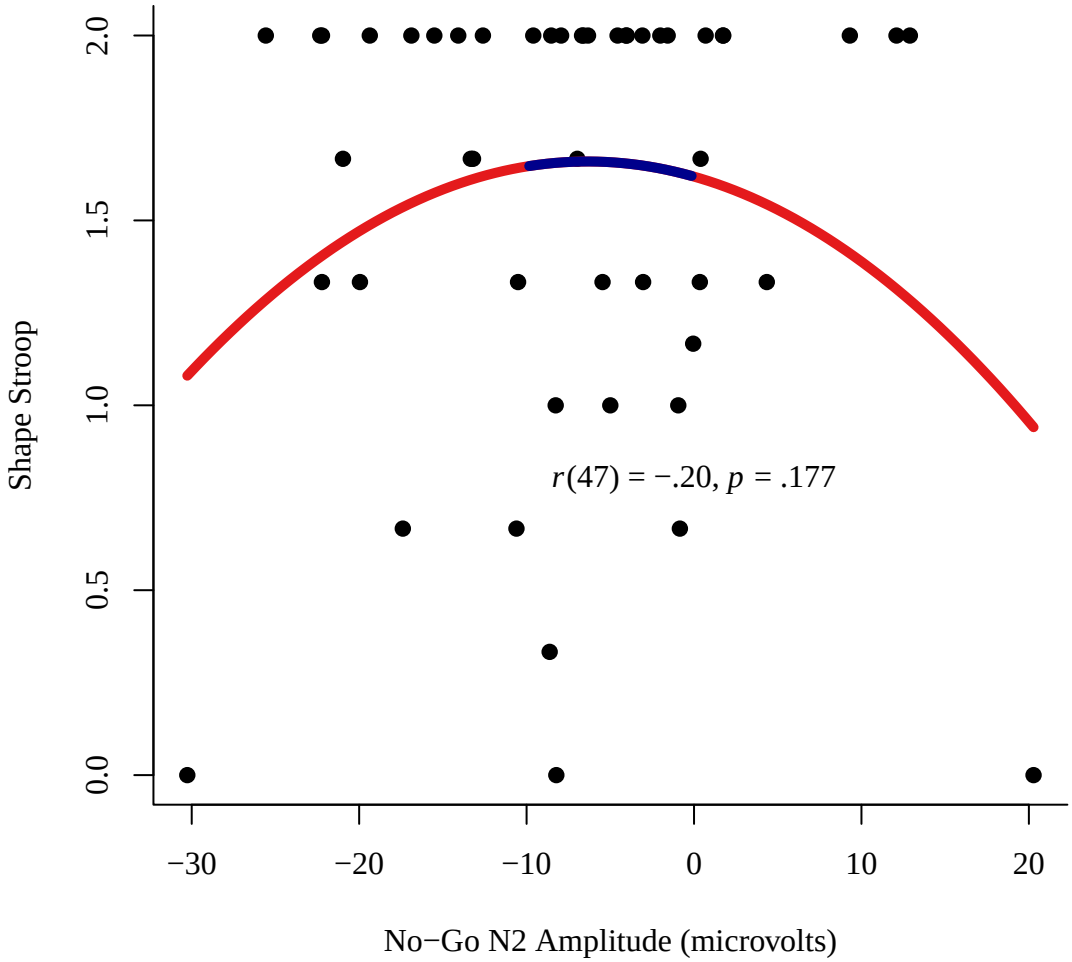

FIGURE 3.19: Quadratic association between no-go N2 amplitude and performance on Shape Stroop. Although the x-axis reflects no-go N2 amplitudes in microvolts, the correlation coefficient reflects the association with no-go N2 amplitudes in *squared* microvolts. Curvilinear fit represents best fitting quadratic form, with blue line from (-10 to 0 μV) and red lines below -10 μV and above 0 μV.

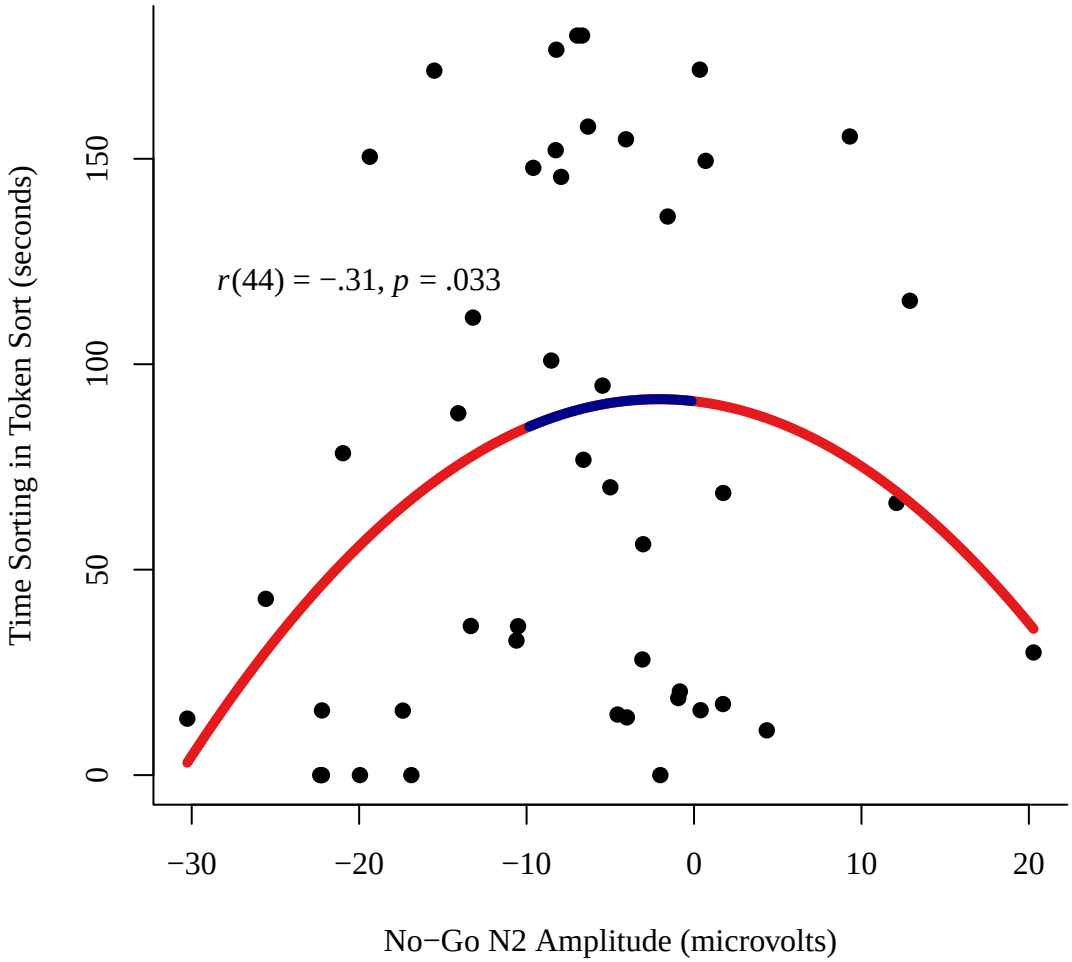

FIGURE 3.20: Quadratic association between no-go N2 amplitude and time sorting in the Token Sort task. Although the x-axis reflects no-go N2 amplitudes in microvolts, the correlation coefficient reflects the association with no-go N2 amplitudes in *squared* microvolts. Curvilinear fit represents best fitting quadratic form, with blue line from (−10 to 0 μV) and red lines below −10 μV and above 0 μV.

TABLE 3.7: Study 2: Pearson Correlations of Children's ERP Components with their Self-Regulation (Lagged).

	P3a					N2				
	Tgt Amp	Frq Amp	Amp Diff	Tgt Lat	Frq Lat	Go Amp	No-Go Amp	Amp Diff	Go Lat	No-Go Lat
Bird/Alligator	−.09	.09	−.16	−.07	.22	−.13	.27	.32	.14	−.11
Shape Stroop	.32	.44*	−.11	.05	.05	.14	−.12	−.22	−.37	.02
Grass/Snow	−.12	.31	−.37†	−.23	−.41*	.27	.19	−.11	−.40	.43†
Token Sort	.19	−.07	.22	−.61**	−.04	.19	−.38	−.45†	−.08	−.04
Sustained Play Attention	−.10	−.43	.60	−.23	.90*	−.63	−.81	−.22	.72	−.66
Fish/Sharks	.10	.48*	−.38	.22	−.03	−.40	.01	.36	.25	−.74**

Note. "Amp" = amplitude, "Lat" = latencies, "Diff" = difference, "Tgt" = target, "Frq" = frequent. Amplitudes are in microvolts, latencies are in milliseconds. P3a amplitude difference reflects target P3a amplitude − frequent P3a amplitude. N2 amplitude difference reflects no-go N2 amplitude − go N2 amplitude. Correlations are two-tailed.

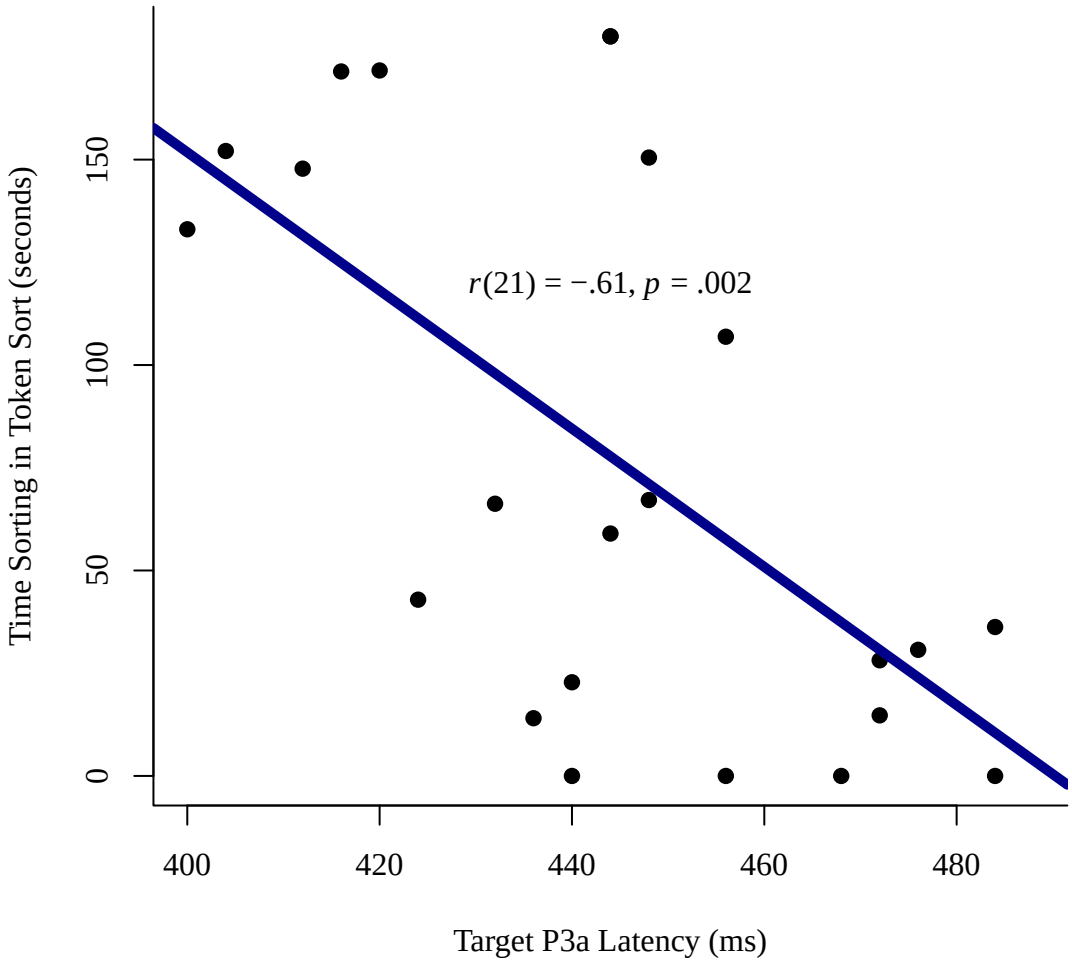

FIGURE 3.21: Association between target P3a latencies and later time sorting on Token Sort.

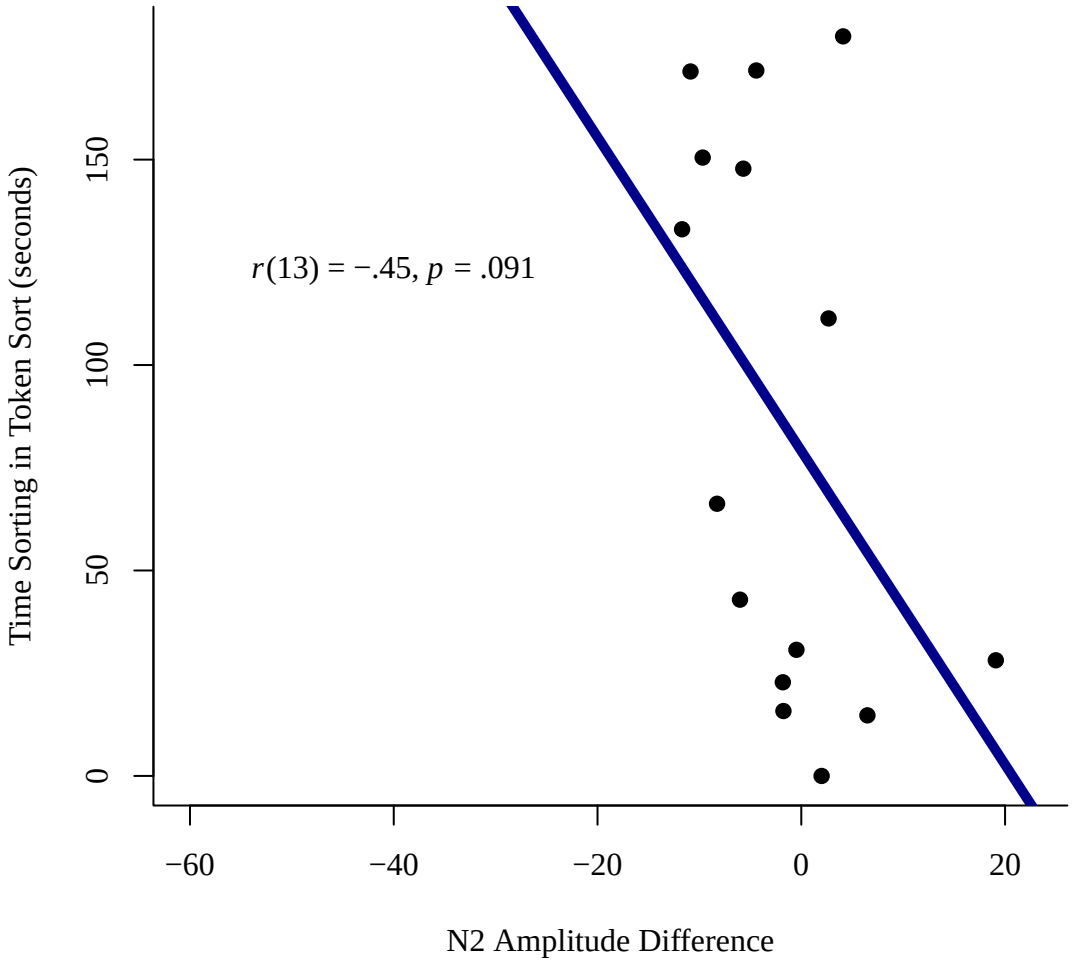

FIGURE 3.22: Association between N2 amplitude difference scores and later time sorting on Token Sort.

Table 3.8: Study 2: Clustered Regression Examining Association of P3a Latencies and N2 Amplitude Difference with Later Time Sorting in Token Sort.

	Dependent variable:	
	Token Sort (lagged)	
Intercept	−72.202 (330.637)	−583.441*** (77.877)
Target P3a Latency	−0.687 (0.560)	
N2 Amplitude Difference		−3.187*** (0.413)
Sex	−32.656 (27.729)	−59.655*** (10.498)
Age	149.588** (46.091)	196.580*** (23.468)
Number of Bad Channels	5.359* (2.369)	9.823***
Number of Target Trials Kept	−1.825 (1.166)	
Number of No-Go Trials Kept		−11.026*** (2.146)
Behavioral Percent Correct on No-Go Trials		1.220* (0.558)
Token Sort	0.668*** (0.152)	0.929*** (0.091)
Observations	19	10
R^2	0.730	0.961
Adjusted R^2	0.596	0.825

Note. $^{\dagger}p < .10$. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$. In the model with N2 amplitude difference scores, the erratic parameter estimates may owe to the small number of remaining degrees of freedom (2) because of the number of predictors (7) given the few number of observations (10). Nevertheless, the association between N2 amplitude difference scores and later Token Sort held over autoregressive controls in models with fewer covariates.

remained associated with performance on Fish/Sharks when examining Spearman's rho ($p = .004$) and after accounting for the nesting of longitudinal data, controlling for prior levels of performance on Fish/Sharks, and controlling for covariates (see Table 3.9).

TABLE 3.9: Study 2: Clustered Regression Examining Association Between No-Go N2 Latencies and Later Performance on Fish/Sharks.

	Dependent variable:
	Behavioral Percent Correct on No-Go Trials (lagged)
Intercept	178.512^{***} (32.863)
No-Go N2 Latency	-0.221^{***} (0.058)
Sex	2.716 (4.004)
Age	−3.597 (6.420)
Number of Bad Channels	−0.292 (0.437)
Number of No-Go Trials Kept	-0.819^{*} (0.354)
Behavioral Percent Correct on No-Go Trials	0.296^{*} (0.131)
Observations	12
R^2	0.692
Adjusted R^2	0.323

Note. $^{\dagger}p < .10$. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

No associations of N2/P3a amplitudes with later self-regulation showed better quadratic than linear fit. A few associations between ERPs and later self-regulation were inconsistent with hypotheses. Longer no-go N2 latencies and smaller P3a amplitude difference scores were marginally significantly associated with better performance on Grass/Snow.

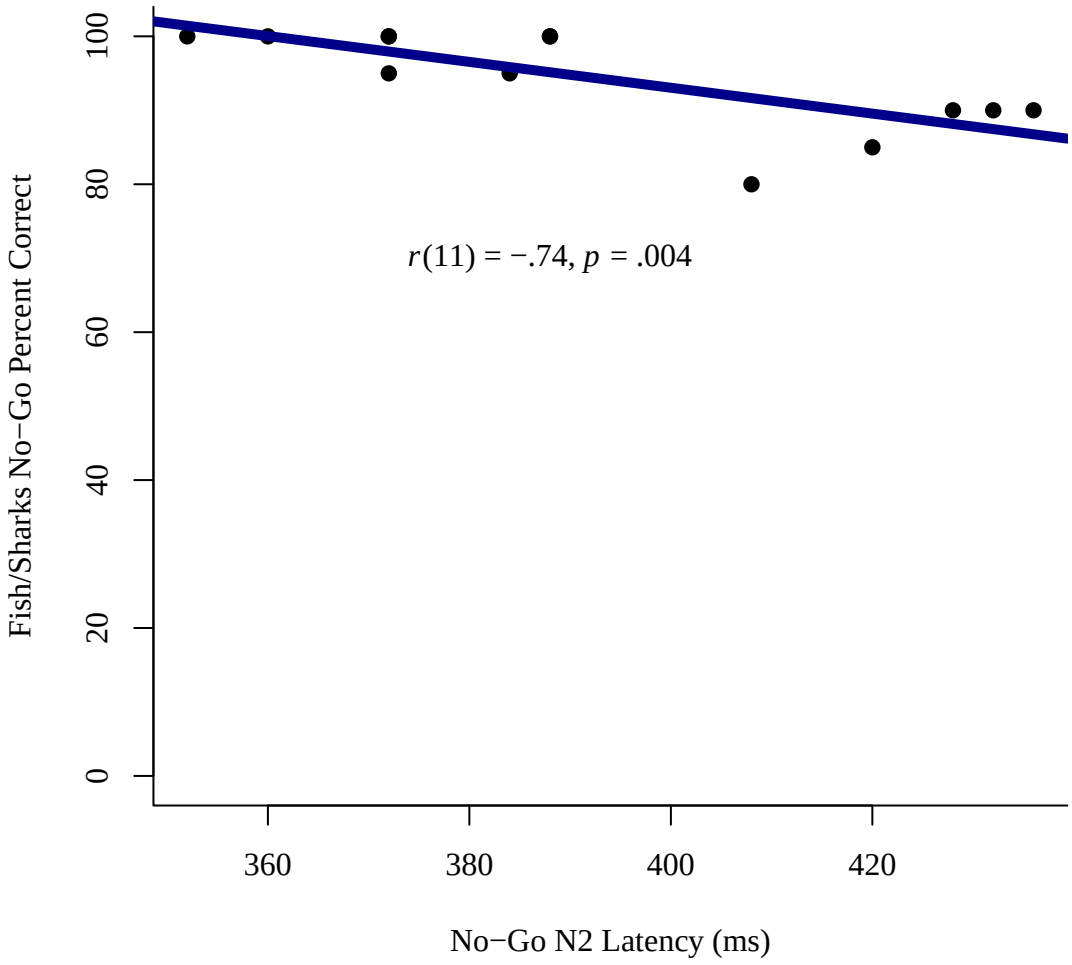

FIGURE 3.23: Association between N2 latencies and later performance on the no-go trials of Fish/Sharks.

3.2.1.0.2 EEG and Self-Regulation. Pearson correlations of children's EEG power values and asymmetry scores with their concurrent self-regulation are in Table 3.10. Consistent with hypotheses, left frontal asymmetry in the Fish/Sharks task was marginally significantly associated with poorer performance on Grass/Snow ($r[44] = -.26$, $p = .080$, see Figure 3.25), and left frontal asymmetry in the oddball task was marginally significantly associated with poorer performance on Sustained Play Attention ($r[44] = -.29$, $p = .052$, see Figure 3.24). Left frontal asymmetry in the Fish/Sharks task remained associated with Grass/Snow when examining Spearman's rho ($p = .071$), but did not remain associated after accounting for the nesting of longitudinal data and controlling for covariates (see Table 3.11). Left frontal asymmetry in the oddball task did not remain associated with Sustained Play Attention when examining Spearman's rho ($p = .276$), but did remain associated after accounting for the nesting of longitudinal data and controlling for covariates (see Table 3.11).

A pattern of associations with frontal alpha power emerged that was inconsistent with hypotheses. More frontal alpha power in the oddball ($r[72] = -.23$, $p = .049$, see Figure 3.26) and Fish/Sharks ($r[44] = -.49$, $p < .001$, see Figure 3.27) tasks was associated with poorer performance on Token Sort. Frontal alpha power in the oddball task did not remain associated with Token Sort when examining Spearman's rho ($p = .162$), suggesting that the association may have owed, in part, to outliers, but did remain associated after accounting for the nesting of longitudinal data and controlling for covariates (see Table 3.12). Frontal alpha power in the Fish/Sharks task remained associated with Token Sort when examining Spearman's rho ($p < .001$), and remained associated after accounting for the nesting of longitudinal data and controlling for covariates (see Table 3.12).

TABLE 3.10: Study 2: Pearson Correlations of Children's EEG and Time-Frequency Components with their Self-Regulation.

	Oddball			Fish/Sharks		
	Frontal Power	Frontal Asymmetry	Frontal TF	Frontal Power	Frontal Asymmetry	Frontal TF
Bird/Alligator	−.02	−.11	.03	−.20	−.01	−.07
Shape Stroop	−.01	−.04	.04	−.15	−.08	.21
Grass/Snow	−.09	.01	.13	−.19	$-.26^{\dagger}$	.05
Token Sort	$-.23^{*}$	.00	.03	$-.49^{**}$	.01	$-.27^{\dagger}$
Sustained Play Attention	.06	$-.29^{\dagger}$	−.21	−.22	.05	−.07
Fish/Sharks	.07	−.06	.15	−.16	−.05	.17

Note. "TF" = time-frequency activity corresponding to timing of P3a (oddball) or N2 (Fish/Sharks), with values in decibels. Power values were log-transformed. Frontal power and asymmetry in alpha frequency range. Frontal time-frequency in theta frequency range. Frontal asymmetry reflects right frontal alpha power − left frontal alpha power (i.e., higher values reflect left frontal asymmetry in alpha frequency range). Correlations are two-tailed.

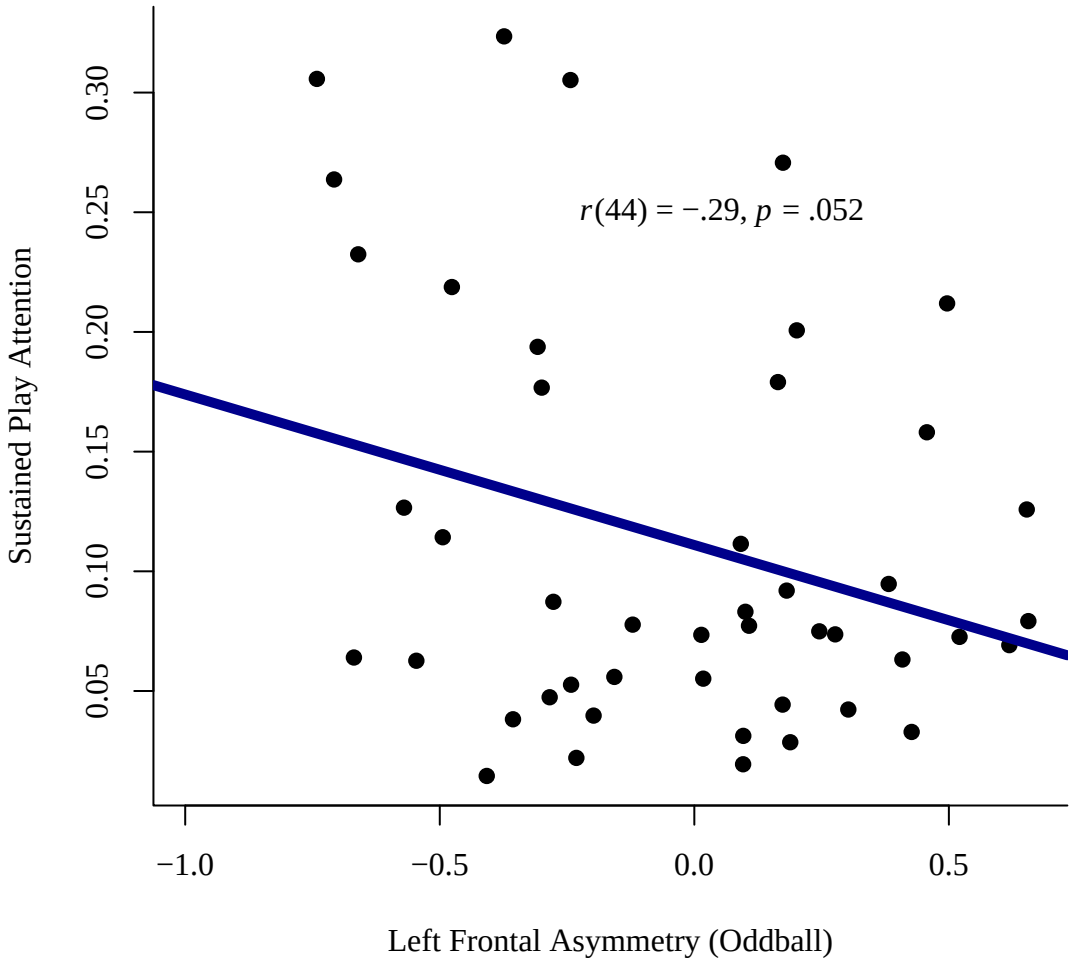

FIGURE 3.24: Association between left frontal asymmetry during the oddball task and performance on Sustained Play Attention.

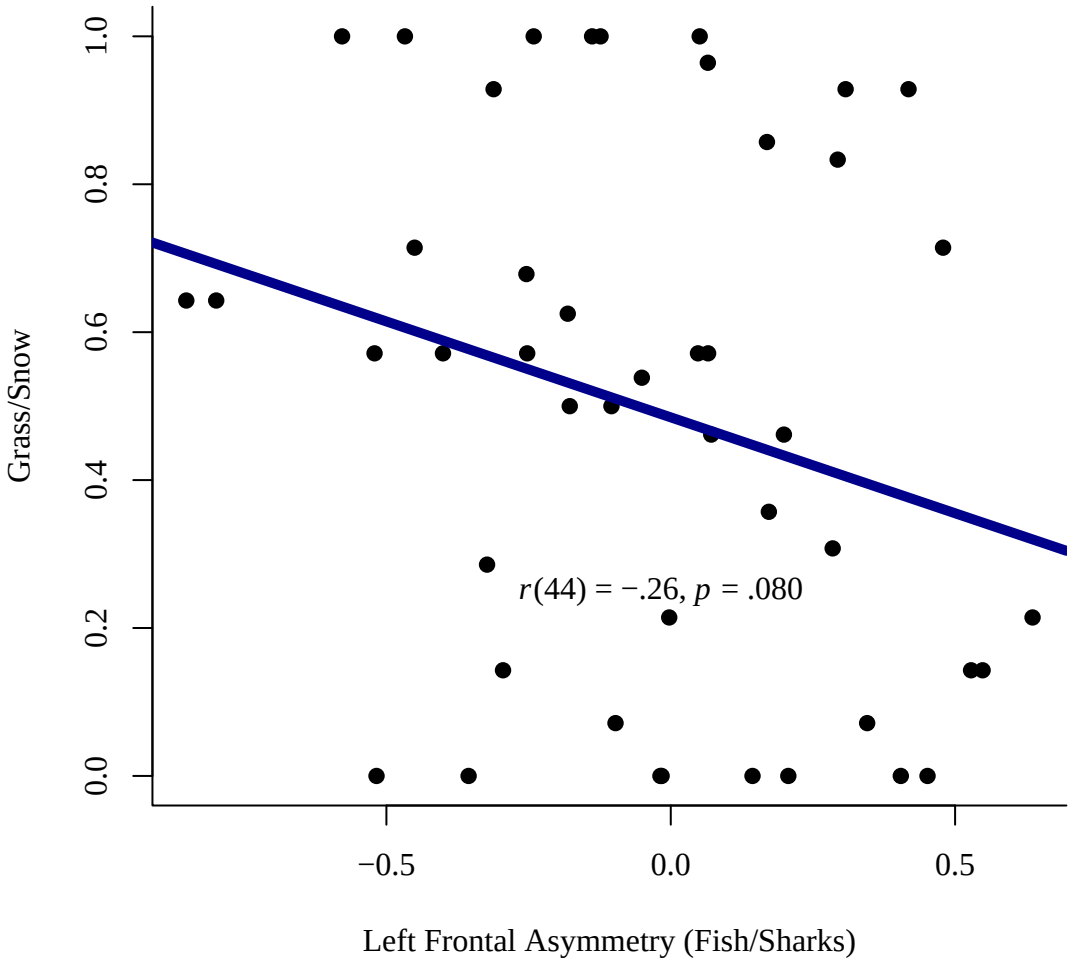

FIGURE 3.25: Association between left frontal asymmetry during the Fish/Sharks task and performance on Grass/Snow.

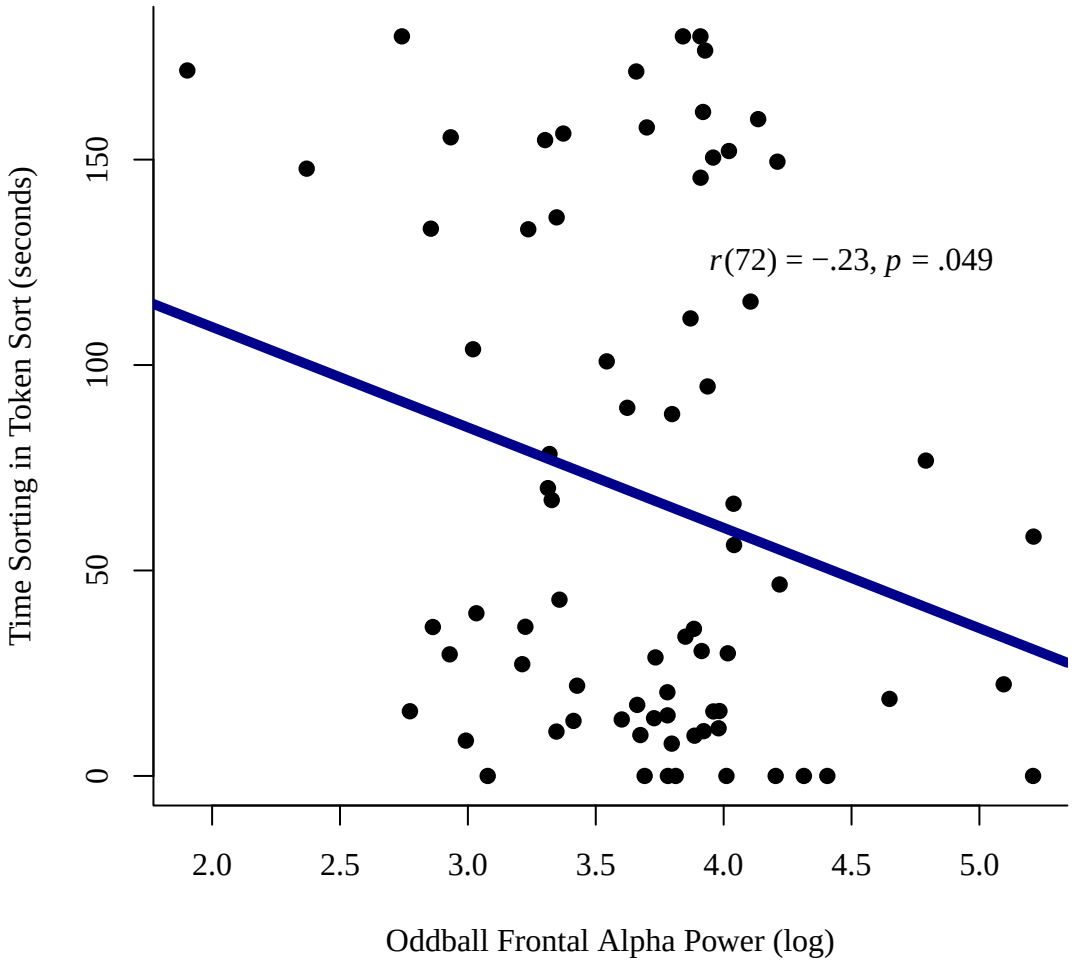

FIGURE 3.26: Association between frontal alpha power during the oddball task and time sorting in Token Sort.

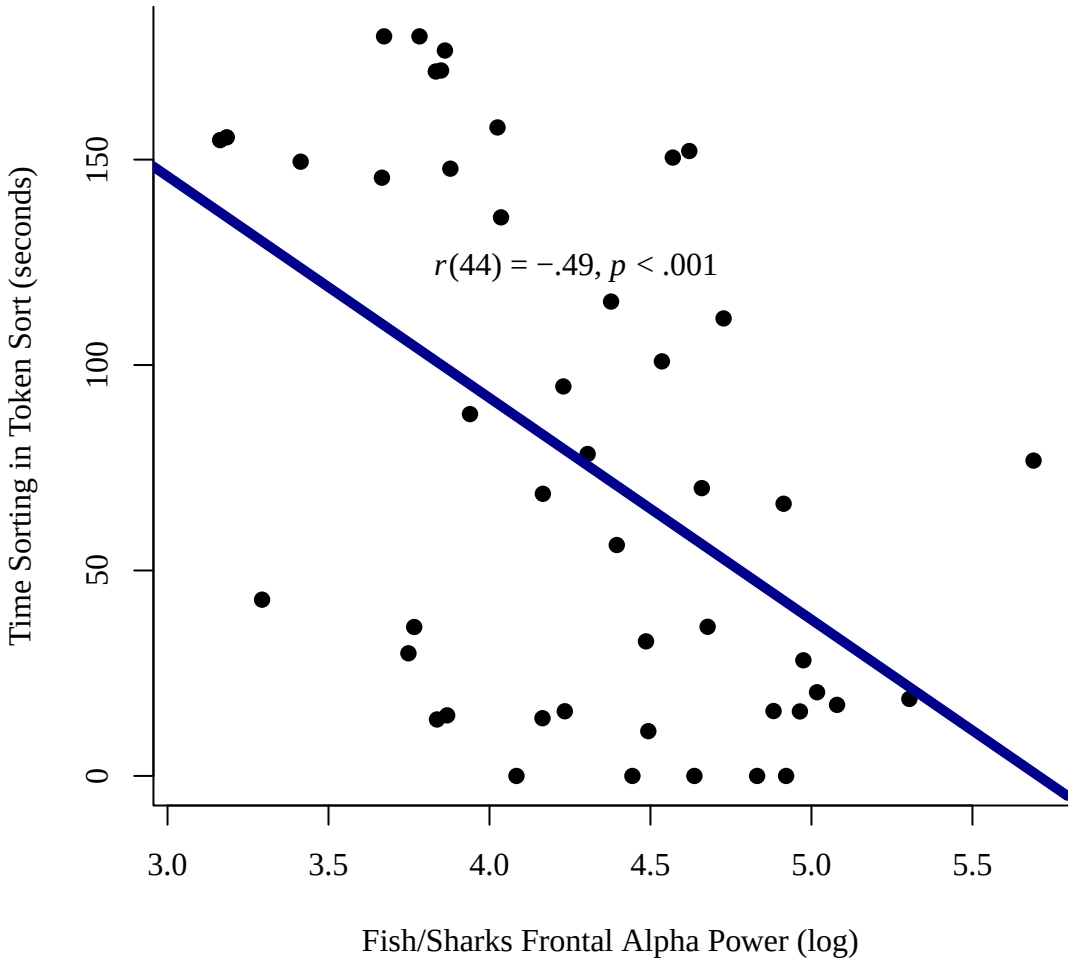

FIGURE 3.27: Association between frontal alpha power during the Fish/Sharks task and time sorting in Token Sort.

Table 3.11: Study 2: Clustered Regression Examining Association Between Left Frontal Asymmetry and Performance on Sustained Play Attention and Grass/Snow.

	Dependent variable:	
	Sustained Play Attention	Grass/Snow
Intercept	−0.064 (0.143)	0.440 (0.534)
Left Frontal Asymmetry (Oddball)	$-0.062^{\dagger}$ (0.033)	
Left Frontal Asymmetry (Fish/Sharks)		−0.074 (0.141)
Sex	0.017 (0.027)	0.178 (0.111)
Age	$0.059^{\dagger}$ (0.033)	0.128 (0.111)
Number of Bad Channels	0.002	-0.051^{**} (0.016)
Number of Target Trials Kept	−0.001 (0.003)	
Number of No-Go Trials Kept		0.007 (0.019)
Behavioral Percent Correct on No-Go Trials		0.001 (0.004)
Observations	45	44
R^2	0.179	0.416
Adjusted R^2	0.074	0.321

Note. $^{\dagger}p < .10$. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

Table 3.12: Study 2: Clustered Regression Examining Association Between Frontal Alpha Power and Performance on Token Sort.

	Dependent variable:	
	Token Sort	
Intercept	−68.683 (83.233)	$243.606^{\dagger}$ (124.928)
Frontal Alpha Power (Oddball)	-24.055^{**} (9.234)	
Frontal Alpha Power (Fish/Sharks)		-46.537^{*} (18.880)
Sex	6.771 (13.866)	11.890 (15.737)
Age	67.212^{***} (13.584)	$35.080^{\dagger}$ (19.910)
Number of Bad Channels	2.115	−2.018 (3.235)
Number of Target Trials Kept	0.102 (1.738)	
Number of No-Go Trials Kept		−2.805 (3.361)
Behavioral Percent Correct on No-Go Trials		−0.231 (0.698)
Observations	71	44
R^2	0.274	0.293
Adjusted R^2	0.218	0.179

Note. $^{\dagger}p < .10$. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

Pearson correlations of children's EEG power values and asymmetry scores with their later self-regulation are in Table 3.13. Consistent with hypotheses, left frontal asymmetry in the Fish/Sharks task was associated with poorer subsequent performance on Sustained Play Attention, but this was only based on 3 cases with later Sustained Play Attention scores, so we prefer not to interpret it in the absence of a pattern of effects.

Inconsistent with hypotheses, however, more frontal alpha power in both the oddball ($r[21] = -.37$, $p = .079$, see Figure 3.28) and Fish/Sharks ($r[13] = -.55$, $p = .035$, see Figure 3.29) tasks was associated with poorer subsequent performance on Token Sort. Frontal alpha power in the oddball task did not remain associated with Token Sort when examining Spearman's rho ($p = .155$) or when accounting for the nesting of longitudinal data, controlling for covariates, and controlling for prior levels of performance on Token Sort (see Table 3.14). Frontal alpha power in the Fish/Sharks task remained associated with Token Sort when examining Spearman's rho ($p = .081$), but did not remain associated after accounting for the nesting of longitudinal data, controlling for covariates, and controlling for prior levels of performance on Token Sort (see Table 3.14).

3.2.1.0.3 Time-Frequency Neurophysiology and Self-Regulation. Pearson correlations of children's time-frequency values with their concurrent self-regulation are in Table 3.10. No concurrent associations between time-frequency values and self-regulation were consistent with hypotheses. Inconsistent with hypotheses, more N2-related frontal theta activity was associated with poorer performance on Token Sort.

Pearson correlations of children's time-frequency values with their later self-regulation are in Table 3.13. Consistent with hypotheses, more N2-related frontal theta activity was

TABLE 3.13: Study 2: Pearson Correlations of Children's EEG and Time-Frequency Components with their Self-Regulation (Lagged).

	Oddball			Fish/Sharks		
	Frontal Power	Frontal Asymmetry	Frontal TF	Frontal Power	Frontal Asymmetry	Frontal TF
Bird/Alligator	−.23	.12	−.09	−.20	−.20	.37
Shape Stroop	−.06	−.11	.30	.08	.11	.41†
Grass/Snow	−.22	.10	−.09	−.39	−.32	.13
Token Sort	−.37†	.29	−.16	−.55*	.25	.09
Sustained Play Attention	−.57	−.04	.54	.56	−1.00†	−.38
Fish/Sharks	−.10	−.27	.34	−.01	.37	.11

Note. "TF" = time-frequency activity corresponding to timing of P3a (oddball) or N2 (Fish/Sharks), with values in decibels. Power values were log-transformed. Frontal power and asymmetry in alpha frequency range. Frontal time-frequency in theta frequency range. Frontal asymmetry reflects right frontal alpha power − left frontal alpha power (i.e., higher values reflect left frontal asymmetry in alpha frequency range). Correlations are two-tailed.

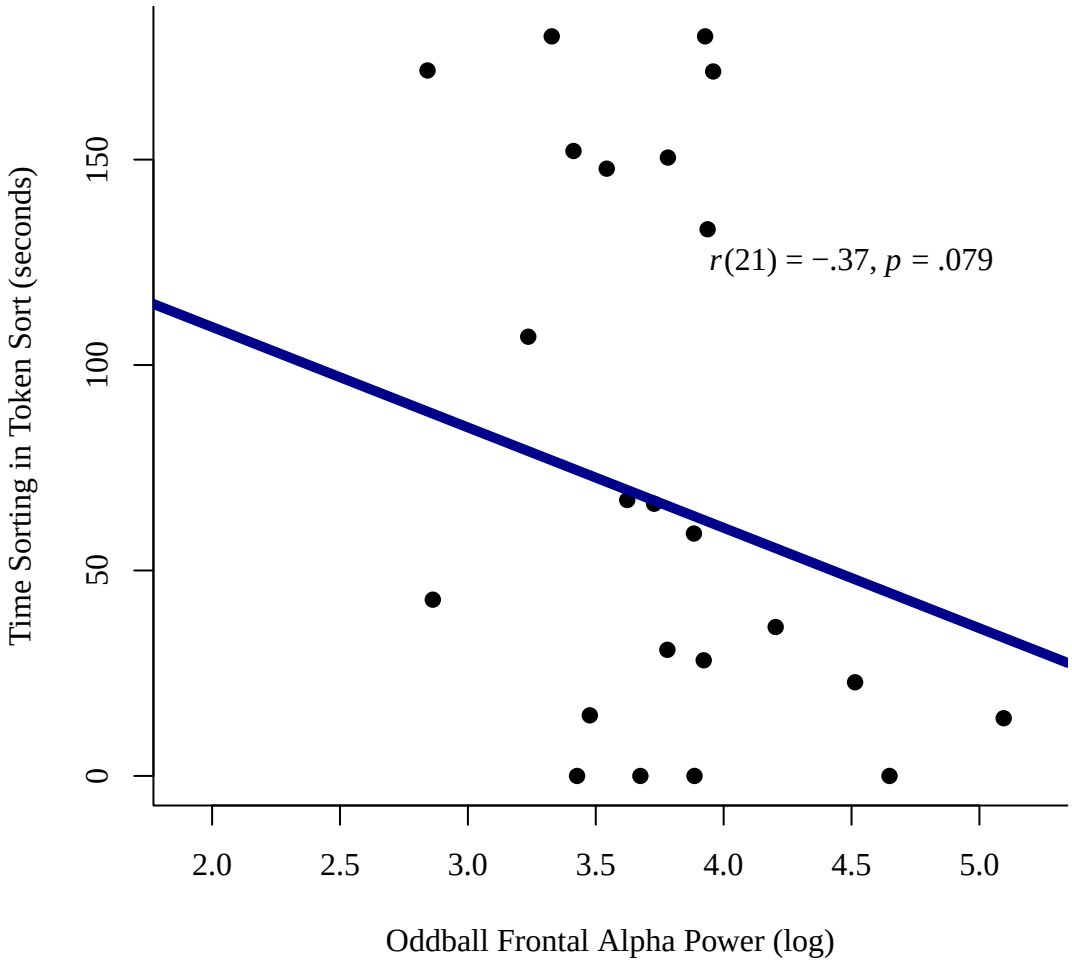

FIGURE 3.28: Association between frontal alpha power during the oddball task and later time sorting in Token Sort.

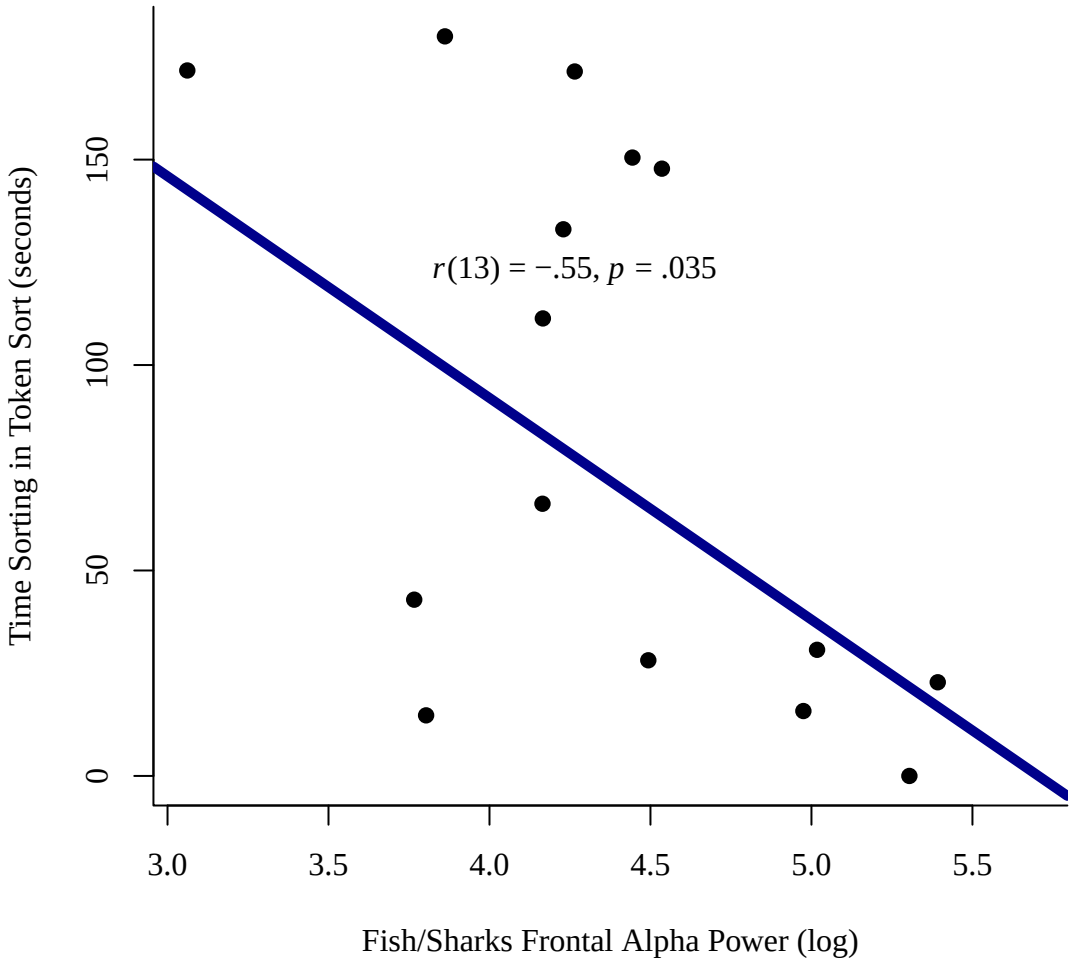

FIGURE 3.29: Association between frontal alpha power during the Fish/Sharks task and later time sorting in Token Sort.

TABLE 3.14: Study 2: Clustered Regression Examining Association Between Frontal Alpha Power and Later Performance on Token Sort.

	Dependent variable:	
	Lagged Token Sort	
Intercept	$-460.338^{\dagger}$ (249.668)	−468.054 (490.938)
Frontal Alpha Power (Oddball)	−0.825 (27.018)	
Frontal Alpha Power (Fish/Sharks)		−8.292 (44.158)
Sex	−13.394 (26.790)	−52.803 (39.744)
Age	168.606^{**} (55.549)	199.865^{*} (99.906)
Number of Bad Channels	6.288^{*}	7.217 (5.894)
Number of Target Trials Kept	−1.124 (2.030)	
Number of No-Go Trials Kept		$-8.536^{\dagger}$ (4.853)
Behavioral Percent Correct on No-Go Trials		0.216 (1.470)
Token Sort	0.763^{***} (0.118)	0.922^{*} (0.441)
Observations	19	10
R^2	0.695	0.822
Adjusted R^2	0.543	0.197

Note. $^{\dagger}p < .10$. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

marginally significantly associated with better subsequent performance on Shape Stroop ($r[16] = .41$, $p = .093$, see Figure 3.30). N2-related frontal theta activity did not remain associated with Shape Stroop when examining Spearman's rho ($p = .147$), but did remain associated when accounting for the nesting of longitudinal data, controlling for covariates, and controlling for prior levels of performance on Shape Stroop (see Table 3.15).

TABLE 3.15: Study 2: Clustered Regression Examining Association Between N2-Related Frontal Theta Activity and Later Performance on Shape Stroop.

	Dependent variable:
	Lagged Shape Stroop
Intercept	1.484* (0.634)
N2-Related Frontal Theta Activity	0.038* (0.016)
Sex	0.311† (0.165)
Age	−0.388* (0.155)
Number of Bad Channels	0.029† (0.016)
Number of No-Go Trials Kept	0.051* (0.022)
Behavioral Percent Correct on No-Go Trials	0.004 (0.005)
Shape Stroop	0.086 (0.078)
Observations	13
R^2	0.470
Adjusted R^2	-0.273

Note. $^{\dagger}p < .10$. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

3.2.2 Association Between Neurophysiology and Externalizing Problems

3.2.2.0.1 ERPs and Externalizing Problems. Pearson correlations of children's ERP components with their concurrent externalizing problems are in Table 3.16. Several associations between ERPs and concurrent externalizing problems were consistent with hypotheses. Longer P3a latencies were associated with more parent-reported attention problems ($r[76] = .30$, $p = .007$, see Figure 3.31) and, to a trend-level, with more secondary caregiver-reported externalizing problems ($r[28] = .33$, $p = .071$, see Figure 3.32)

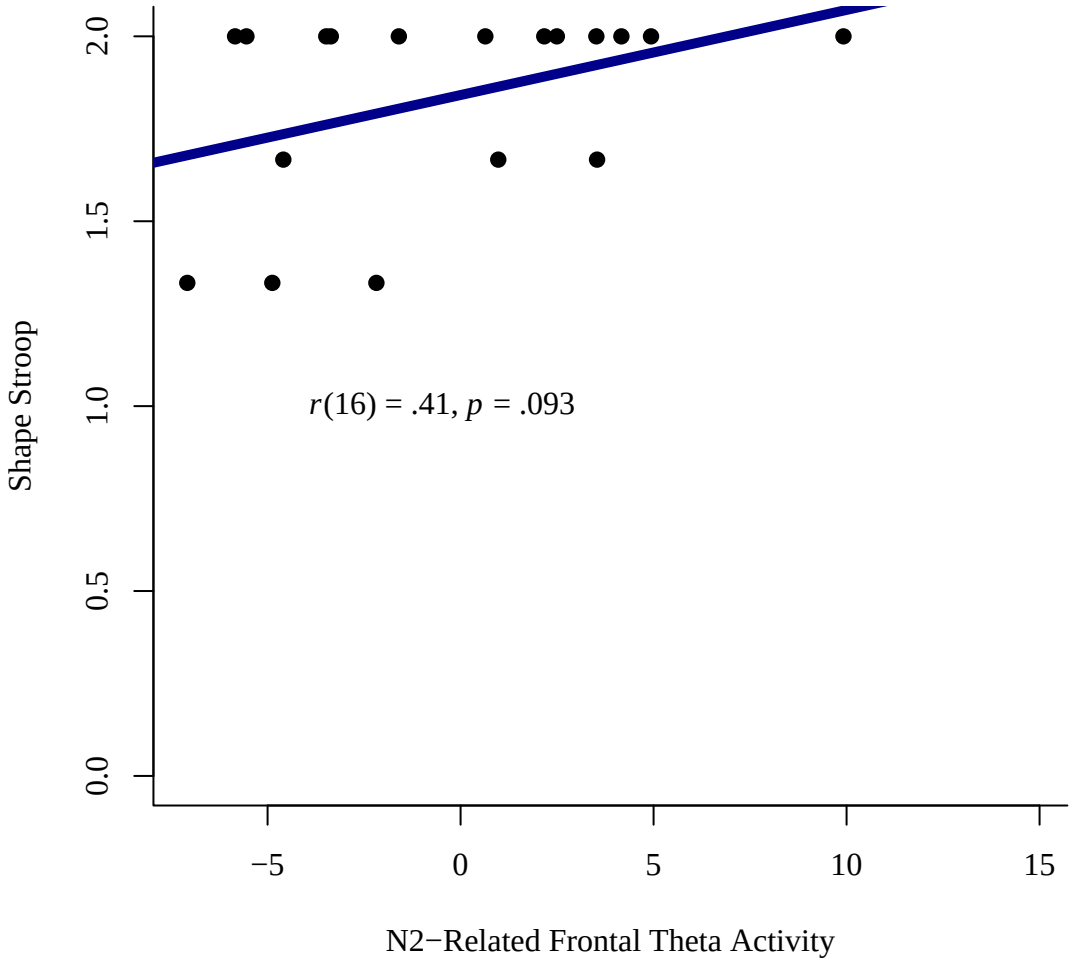

FIGURE 3.30: Association between N2-related frontal theta activity and later performance on Shape Stroop.

and aggression ($r[28] = .31$, $p = .096$, see Figure 3.33). P3a latencies remained associated with externalizing ($p = .086$) and attention problems ($p = .013$), but not with aggression ($p = .176$), when examining Spearman's rho and accounting for the nesting of longitudinal data and controlling for covariates (see Table 3.17).

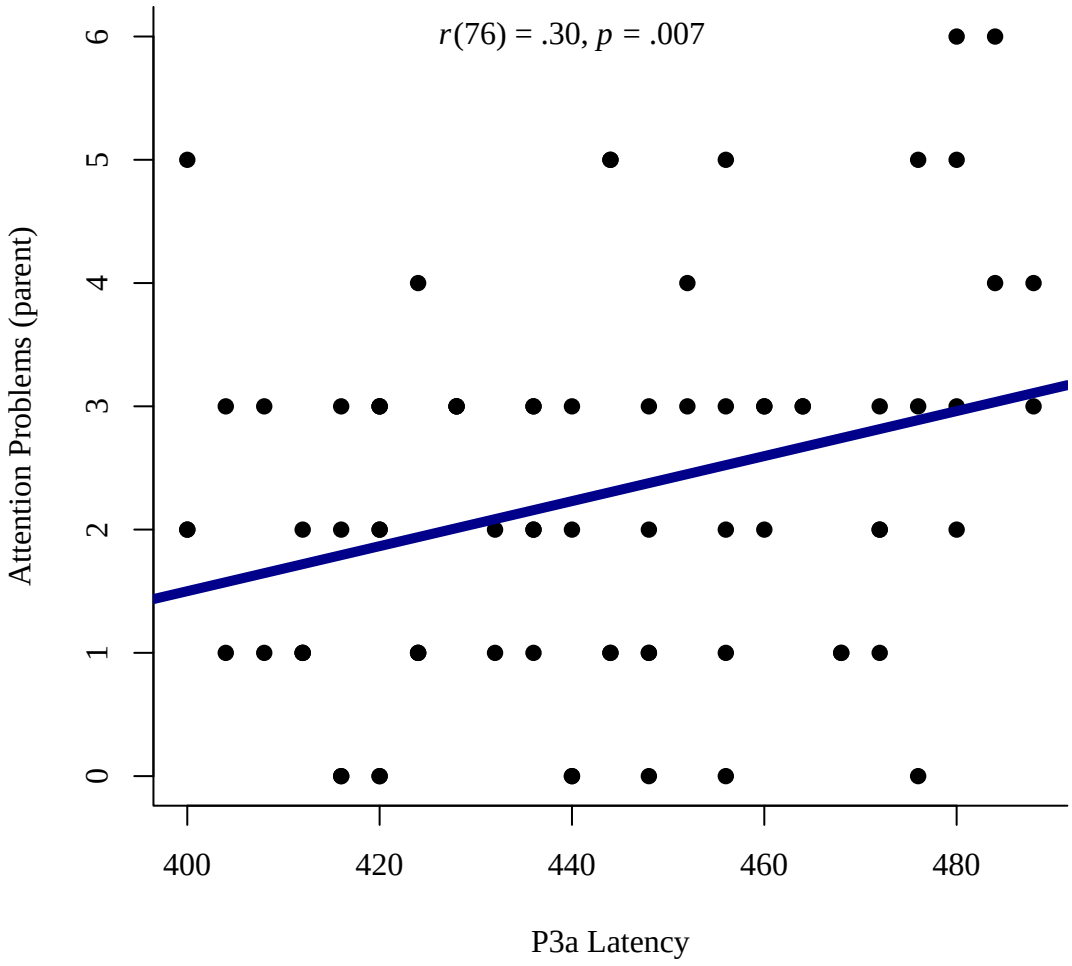

FIGURE 3.31: Association between P3a latencies and parent-reported CBCL Attention Problems.

A few associations between ERPs and externalizing problems were inconsistent with

TABLE 3.16: Study 2: Pearson Correlations of Children's ERP Components with their Externalizing Problems.

	P3a					N2				
	Tgt Amp	Frq Amp	Amp Diff	Tgt Lat	Frq Lat	Go Amp	No-Go Amp	Amp Diff	Go Lat	No-Go Lat
CBCL EXT Parent	−.08	.05	−.11	.09	−.36**	−.04	.09	.11	.23	−.23
CBCL EXT Secondary	−.06	−.25	.19	.33†	−.09	−.63**	−.38†	.11	.31	.22
CBCL AGG Parent	−.07	.04	−.10	.02	−.39**	−.03	.07	.08	.27†	−.24
CBCL AGG Secondary	.00	−.19	.20	.31†	−.12	−.60**	−.29	.17	.44†	.23
CBCL ATT Parent	−.09	.06	−.13	.30**	−.11	−.04	.12	.13	−.06	−.08
CBCL ATT Secondary	−.21	−.32†	.12	.29	.03	−.43†	−.41†	−.07	−.09	.11
ECBI Intensity Parent	.00	−.09	.09	−.09	−.15	−.05	.01	.05	.24	−.20

Note. "Amp" = amplitude, "Lat" = latencies, "Diff" = difference, "Tgt" = target, "Frq" = frequent, "EXT" = externalizing problems, "AGG" = aggression, "ATT" = attention problems. Amplitudes are in microvolts, latencies are in milliseconds. P3a amplitude difference reflects target P3a amplitude − frequent P3a amplitude. N2 amplitude difference reflects no-go N2 amplitude − go N2 amplitude. Correlations are two-tailed.

TABLE 3.17: Study 2: Clustered Regression Examining Association Between P3a Latencies and Externalizing, Aggression, and Attention Problems.

	Dependent variable:		
	Externalizing Problems (secondary caregiver)	Aggression (secondary caregiver)	Attention Problems (parent)
Intercept	−24.946 (21.134)	−12.206 (16.453)	−5.283 (3.220)
P3a Latency	0.073* (0.037)	0.048 (0.030)	0.017* (0.007)
Sex	−1.969 (1.567)	−1.665 (1.344)	−0.300 (0.373)
Age	0.750 (2.169)	−0.333 (1.736)	0.239 (0.349)
Number of Bad Channels	0.099 (0.311)	0.006 (0.270)	0.013 (0.074)
Number of Target Trials Kept	−0.236 (0.154)	−0.208† (0.125)	−0.039 (0.055)
Observations	28	28	75
R^2	0.242	0.198	0.124
Adjusted R^2	0.069	0.016	0.060

Note. $^{\dagger}p < .10$. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

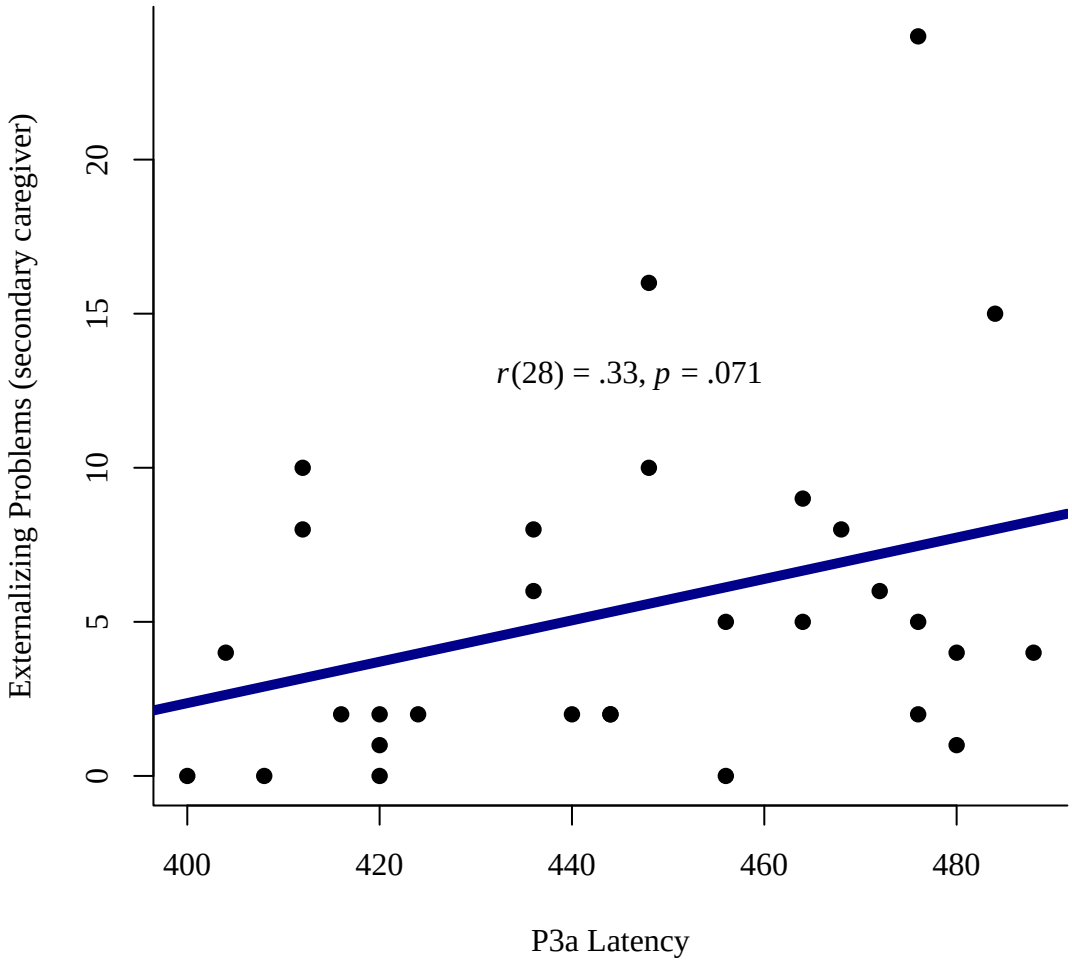

FIGURE 3.32: Association between P3a latencies and secondary caregiver-reported CBCL Externalizing Problems.

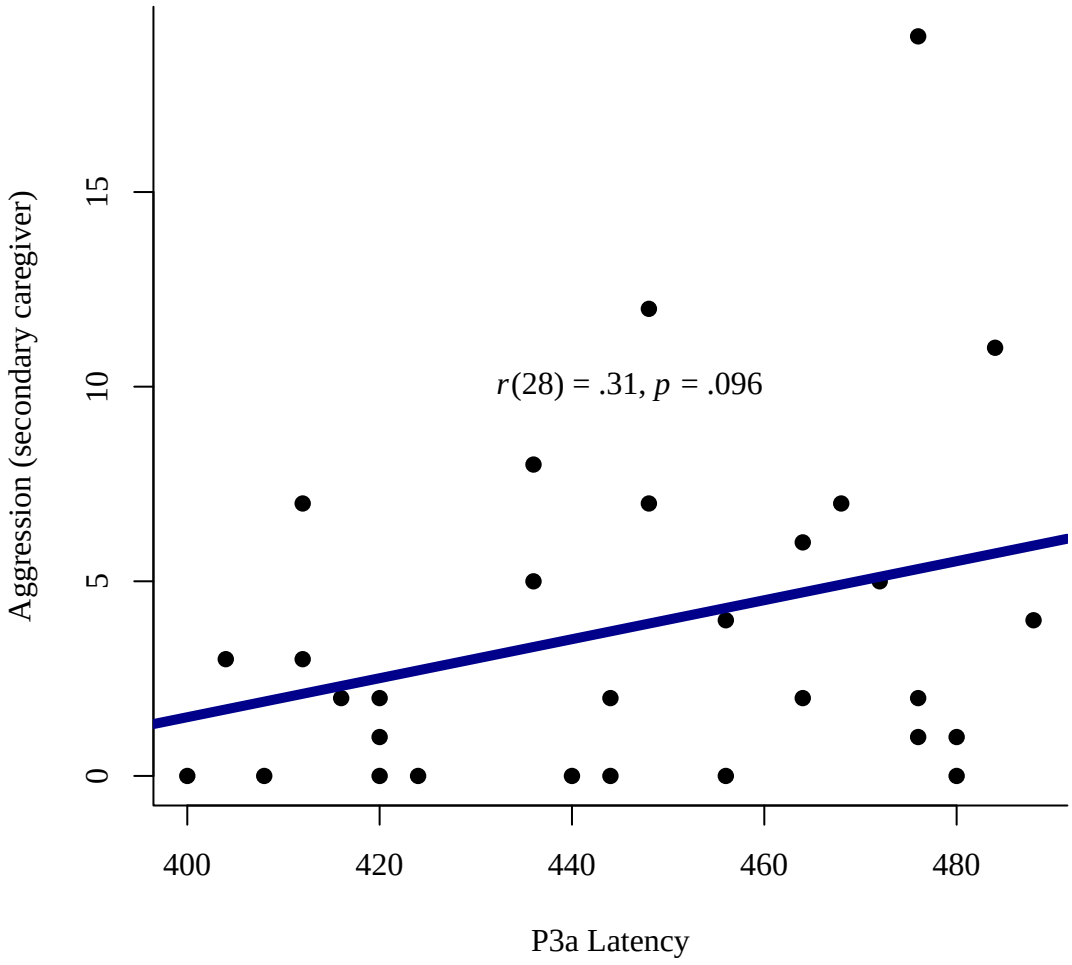

FIGURE 3.33: Association between P3a latencies and secondary caregiver-reported CBCL Aggression.

hypotheses. Larger (more negative) no-go N2 amplitudes were marginally significantly associated with both more secondary caregiver-reported externalizing and more attention problems.

However, the association between no-go N2 amplitudes and secondary caregiver-reported attention problems showed better quadratic than linear fit ($F[1] = 7.74$, $p = .012$), suggesting that the linear association may have masked a nonlinear association. The clustered quadratic regression results of the association between target P3b amplitudes and attention problems are in Table 3.18, and the quadratic association is depicted in Figure 3.34. Examination of the scatterplot (Figure 3.34) suggests that there was a U-shaped association between N2 amplitudes and attention problems. The quadratic association between N2 amplitudes and attention problems remained significant when examining Spearman's rho ($p = .014$) and when accounting for the nesting of longitudinal data and controlling for covariates. Attention problems were lowest among children with middle-range (-10 to 0 μV) N2 amplitudes and were highest among children with larger (< -10 μV) or smaller N2 (> 0 μV) amplitudes. Findings suggest that there may be an optimal range of N2 amplitudes, and children whose N2 amplitudes are excessively large or small may have more attention problems. We present the quadratic association with caution, however, because of the modest sample size of children with reports from secondary caregivers and the possibility of over-fitting owing to the estimation of additional, non-linear parameters in a modest sample (i.e., we might be modeling noise).

Pearson correlations of children's ERP components with their later externalizing problems are in Table 3.19. No associations between ERPs and later externalizing problems were consistent with hypotheses, whereas several associations were inconsistent with hypotheses.

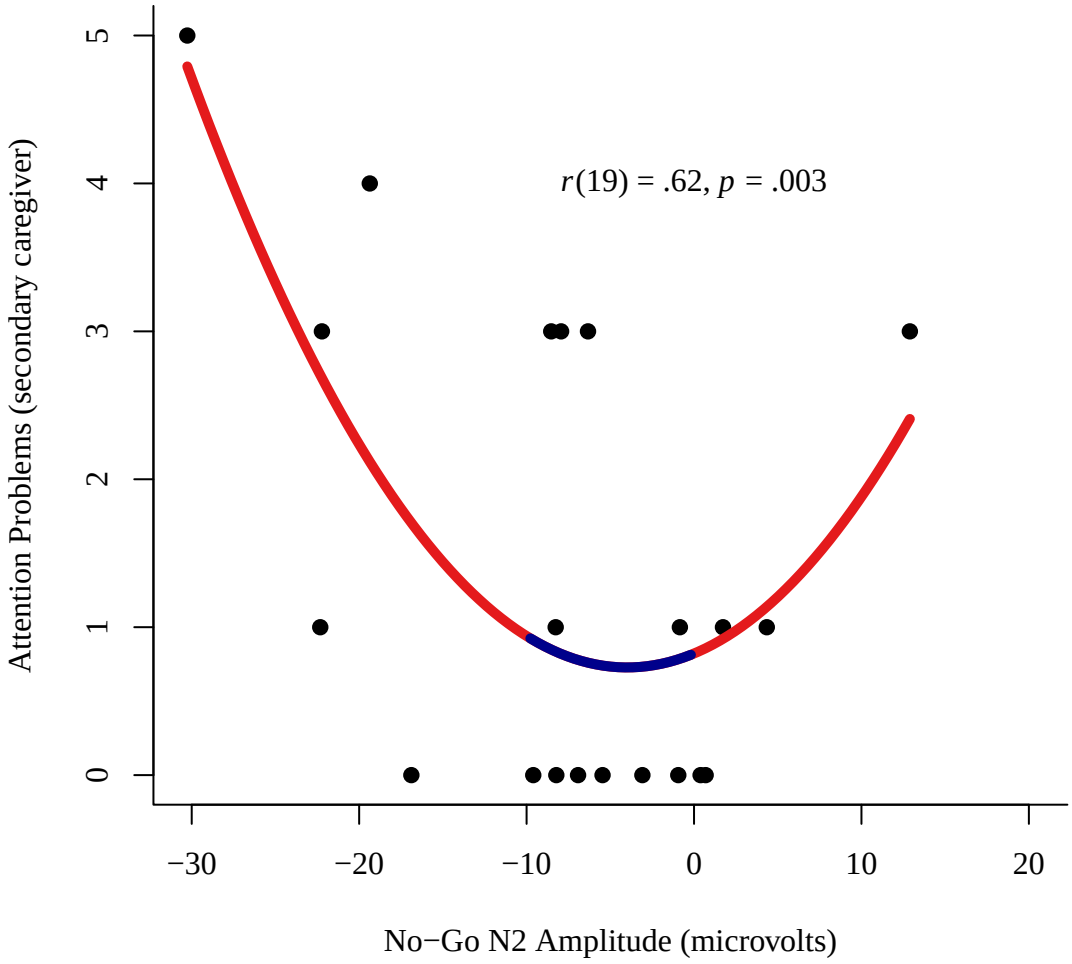

FIGURE 3.34: Quadratic association between no-go N2 amplitude and secondary caregiver-reported attention problems on the CBCL. Although the x-axis reflects no-go N2 amplitudes in microvolts, the correlation coefficient reflects the association with no-go N2 amplitudes in *squared* microvolts. Curvilinear fit represents best fitting quadratic form, with blue line from (-10 to 0 μV) and red lines below -10 μV and above 0 μV.

TABLE 3.18: Study 2: Clustered Linear and Quadratic Regression Examining Association Between N2 Amplitude and Secondary Caregiver-Reported CBCL Attention Problems.

	Dependent variable:	
	CBCL Attention Problems (Secondary)	
	Linear	Quadratic
Intercept	-5.409^{***} (1.536)	-3.338^{*} (1.544)
No-Go N2 Amplitude (Linear)	-0.108^{**} (0.034)	-0.012 (0.041)
No-Go N2 Amplitude (Quadratic)		0.006^{*} (0.002)
Sex	-0.588 (0.538)	0.341 (0.758)
Age	$1.234^{\dagger}$ (0.705)	0.348 (0.780)
Number of Bad Channels	-0.044 (0.141)	-0.184 (0.137)
Number of No-Go Trials Kept	$-0.193^{\dagger}$ (0.110)	-0.121 (0.101)
Behavioral Percent Correct on No-Go Trials	0.061^{*} (0.027)	0.073^{*} (0.032)
Observations	19	19
R^2	0.507	0.627
Adjusted R^2	0.261	0.390

Note. $^{\dagger}p < .10$. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

Larger (more negative) no-go N2 amplitudes were associated with later parent-reported attention problems. Shorter no-go N2 latencies were associated with later secondary caregiver-reported externalizing problems and aggression, but this was based on very few cases with later reports by secondary caregivers (4).

3.2.2.0.2 EEG and Externalizing Problems. Pearson correlations of children's EEG power values and asymmetry scores with their concurrent externalizing problems are in Table 3.20. There were a number of associations between EEG power/asymmetry and concurrent externalizing problems that were consistent with hypotheses. Less frontal alpha power in the oddball task was associated with more parent-reported aggression ($r[77] = -.23$, $p = .040$, see Figure 3.35) and, to a trend-level, with more CBCL externalizing problems ($r[77] = -.20$, $p = .082$, see Figure 3.36) and ECBI behavior problems ($r[77] = -.20$, $p = .083$, see Figure 3.37). Less frontal alpha power in the oddball task remained associated with CBCL aggression ($p = .075$) and ECBI behavior problems ($p = .086$) when examining Spearman's rho, but did not remain significantly associated with CBCL externalizing problems ($p = .125$). Less frontal alpha power in the oddball task remained associated with CBCL aggression and externalizing problems when accounting for the nesting of longitudinal data and controlling for covariates, but the association did not remain when examining ECBI behavior problems (see Table 3.21).

Less frontal alpha power in the Fish/Sharks task was associated with more parent-reported ECBI behavior problems ($r[49] = -.36$, $p = .011$, see Figure 3.38) and CBCL externalizing ($r[49] = -.45$, $p < .001$, see Figure 3.39), aggression ($r[49] = -.43$, $p = .002$, see Figure 3.40), and attention problems ($r[49] = -.28$, $p = .046$, see Figure 3.41). Less

TABLE 3.19: Study 2: Pearson Correlations of Children's ERP Components with their Externalizing Problems (Lagged).

	P3a					N2				
	Tgt Amp	Frq Amp	Amp Diff	Tgt Lat	Frq Lat	Go Amp	No-Go Amp	Amp Diff	Go Lat	No-Go Lat
CBCL EXT Parent	.01	.13	−.10	.01	−.11	.02	−.21	−.18	−.15	−.26
CBCL EXT Secondary	−.38	−.50	.16	.14	.70	−.52	−.63	−.28	.78	−.94†
CBCL AGG Parent	.00	.20	−.17	−.01	−.08	.01	−.16	−.14	−.13	−.24
CBCL AGG Secondary	−.26	−.37	.15	.16	.72	−.50	−.46	−.14	.89	−.99**
CBCL ATT Parent	.04	−.21	.21	.08	−.19	.05	−.41†	−.36	−.23	−.29
CBCL ATT Secondary	−.60	−.68†	.16	.04	.40	−.46	−.88	−.53	.44	−.69
ECBI Intensity Parent	.08	.06	.02	−.08	−.06	−.08	.05	.12	.16	−.32

Note. "Amp" = amplitude, "Lat" = latencies, "Diff" = difference, "Tgt" = target, "Frq" = frequent, "EXT" = externalizing problems, "AGG" = aggression, "ATT" = attention problems. Amplitudes are in microvolts, latencies are in milliseconds. P3a amplitude difference reflects target P3a amplitude − frequent P3a amplitude. N2 amplitude difference reflects no-go N2 amplitude − go N2 amplitude. Correlations are two-tailed.

Table 3.20: Study 2: Pearson Correlations of Children's EEG and Time-Frequency Components with their Externalizing Problems.

	Oddball			Fish/Sharks		
	Frontal Power	Frontal Asymmetry	Frontal TF	Frontal Power	Frontal Asymmetry	Frontal TF
CBCL EXT Parent	$-.20^{\dagger}$	.08	$-.23^{*}$	$-.45^{**}$	.04	$-.05$
CBCL EXT Secondary	$-.06$	.01	.25	.02	$.39^{\dagger}$	$-.29$
CBCL AGG Parent	$-.23^{*}$	.08	$-.24^{*}$	$-.43^{**}$	.03	$-.07$
CBCL AGG Secondary	$-.04$	$-.04$	.24	.14	$.38^{\dagger}$	$-.32$
CBCL ATT Parent	.00	.06	$-.08$	$-.28^{*}$	.03	.04
CBCL ATT Secondary	$-.09$	.13	.21	$-.25$	.24	$-.11$
ECBI Intensity Parent	$-.20^{\dagger}$	$.24^{*}$	$-.15$	$-.36^{*}$	.08	.15

Note. "TF" = time-frequency activity corresponding to timing of P3a (oddball) or N2 (Fish/Sharks), with values in decibels. "EXT" = externalizing problems, "AGG" = aggression, "ATT" = attention problems. Power values were log-transformed. Frontal power and asymmetry in alpha frequency range. Frontal time-frequency in theta frequency range. Frontal asymmetry reflects right frontal alpha power − left frontal alpha power (i.e., higher values reflect left frontal asymmetry in alpha frequency range). Correlations are two-tailed.

TABLE 3.21: Study 2: Clustered Regression Examining Association of Frontal Alpha Power (Oddball) with Aggression and Externalizing Problems.

	Dependent variable:		
	CBCL Aggression (parent)	CBCL Externalizing Problems (parent)	ECBI Behavior Problems (parent)
Intercept	22.329** (7.557)	27.112** (9.180)	150.926*** (35.112)
Frontal Alpha Power (oddball)	−2.920** (0.896)	−3.368*** (0.998)	−7.190 (4.451)
Sex	−1.976 (1.227)	−2.708† (1.459)	−3.174 (5.524)
Age	1.048 (1.326)	1.199 (1.492)	4.104 (4.766)
Number of Bad Channels	−0.154 (0.211)	−0.148 (0.272)	−0.974 (0.793)
Number of Target Trials Kept	−0.169 (0.171)	−0.231 (0.208)	−0.942 (0.810)
Observations	76	76	76
R^2	0.121	0.126	0.081
Adjusted R^2	0.059	0.063	0.015

Note. $^{\dagger}p < .10$. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

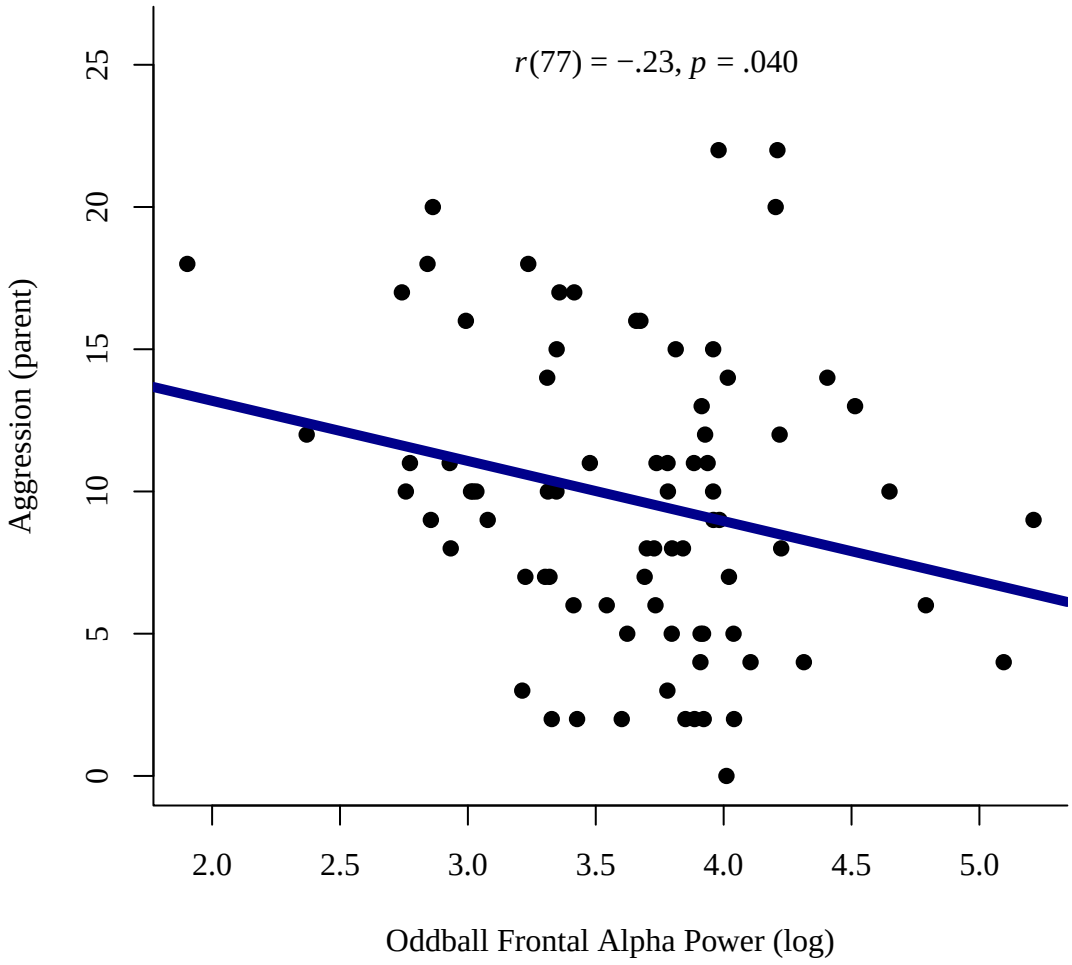

FIGURE 3.35: Association between frontal alpha power during the oddball task and parent-reported CBCL Aggression.

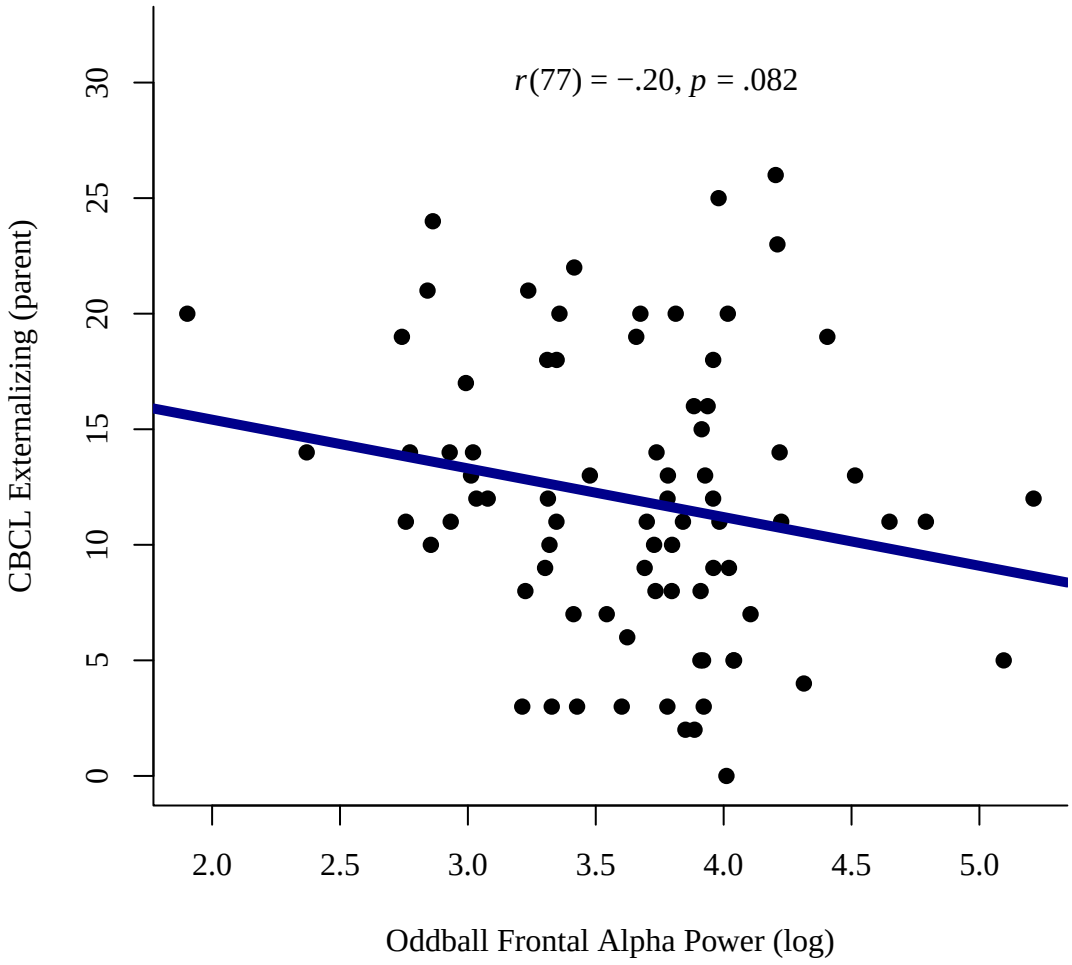

FIGURE 3.36: Association between frontal alpha power during the oddball task and parent-reported CBCL Externalizing Problems.

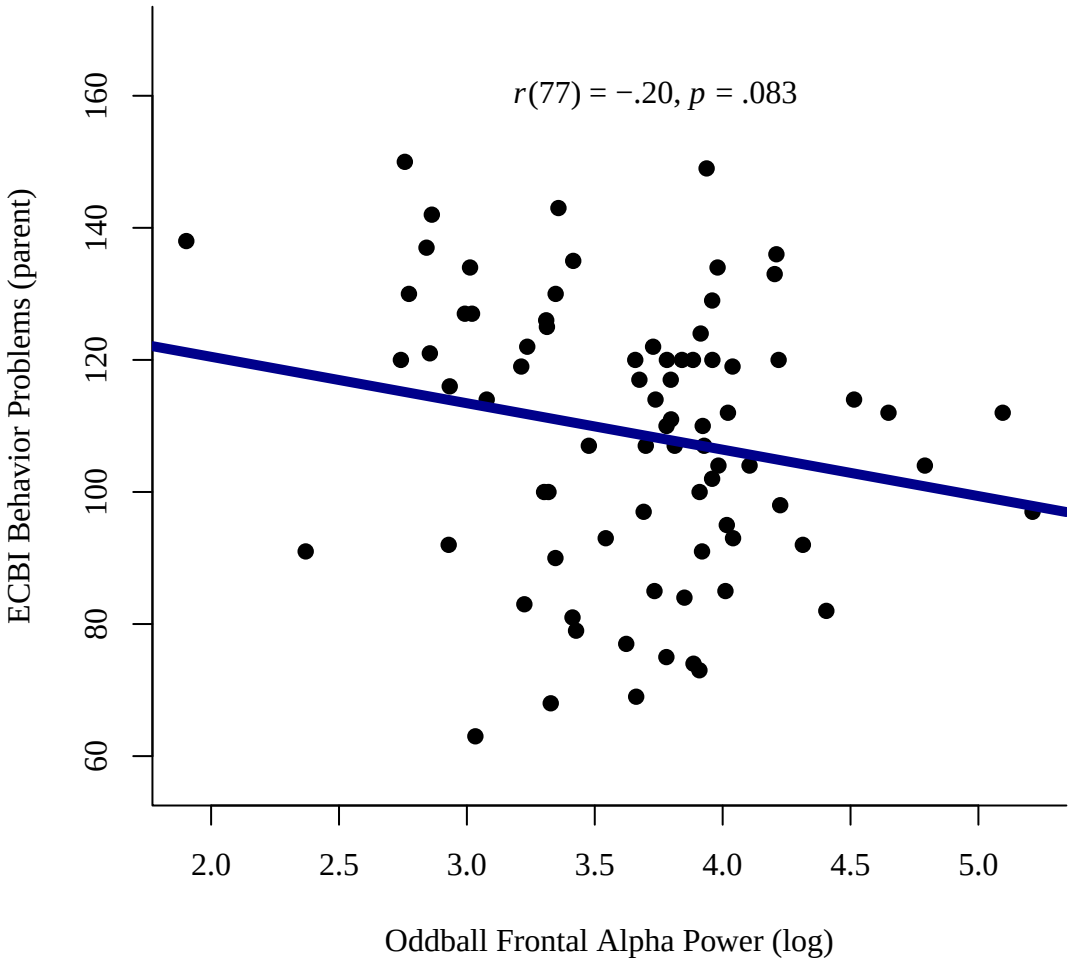

FIGURE 3.37: Association between frontal alpha power during the oddball task and parent-reported intensity of behavior problems on the ECBI.

frontal alpha power in the Fish/Sharks task remained associated with ECBI behavior problems ($p = .015$) and CBCL externalizing ($p < .001$), aggression ($p = .002$), and attention problems ($p = .010$) when examining Spearman's rho. Less frontal alpha power in the Fish/Sharks task remained associated with CBCL externalizing, aggression, and attention problems when accounting for the nesting of longitudinal data and controlling for covariates. However, the same was not so for the association with parent-reported behavior problems on the ECBI (see Table 3.22).

Left frontal asymmetry in the oddball task was associated with more parent-reported ECBI behavior problems ($r[77] = .24$, $p = .035$, see Figure 3.42). Left frontal asymmetry in the oddball task remained associated with ECBI behavior problems when examining Spearman's rho ($p = .030$) and when accounting for the nesting of longitudinal data and controlling for covariates (see Table 3.23). Left frontal asymmetry in the Fish/Sharks task was marginally significantly associated with more secondary caregiver-reported aggression ($r[19] = .38$, $p = .087$, see Figure 3.44) and externalizing problems ($r[19] = .39$, $p = .081$, see Figure 3.43). Left frontal asymmetry in the Fish/Sharks task did not remain associated with aggression ($p = .177$) or externalizing problems ($p = .219$) when examining Spearman's rho or when accounting for the nesting of longitudinal data and controlling for covariates (see Table 3.23), suggesting that the associations may have owed, in part, to outliers.

Pearson correlations of children's EEG power values and asymmetry scores with their later externalizing problems are in Table 3.24. Consistent with hypotheses, less frontal alpha power in the Fish/Sharks task was associated with later parent reported aggression ($r[16] = -.68$, $p = .002$, see Figure 3.45), externalizing problems ($r[16] = -.65$, $p = .003$, see Figure 3.46) and ECBI behavior problems ($r[15] = -.62$, $p = .008$, see Figure 3.47).

TABLE 3.22: Study 2: Clustered Regression Examining Association of Frontal Alpha Power (Fish/Sharks) with Aggression, Attention, and Externalizing Problems.

	Dependent variable:			
	CBCL Aggression (parent)	CBCL Attention Problems (parent)	CBCL Externalizing Problems (parent)	ECBI Behavior Problems (parent)
Intercept	27.542** (8.549)	11.481*** (3.027)	39.023*** (9.743)	177.925*** (46.754)
Frontal Alpha Power (Fish/Sharks)	−2.599* (1.133)	−0.876† (0.488)	−3.475** (1.326)	−6.531 (4.657)
Sex	0.013 (1.401)	−0.653† (0.351)	−0.640 (1.511)	−0.720 (5.000)
Age	−1.466 (1.590)	−0.376 (0.422)	−1.842 (1.744)	−1.886 (6.014)
Number of Bad Channels	−0.466* (0.209)	−0.192*** (0.055)	−0.658** (0.236)	−2.269* (0.959)
Number of No-Go Trials Kept	0.270 (0.246)	−0.188* (0.081)	0.082 (0.293)	0.128 (0.973)
Behavioral Percent on No-Go Trials	−0.004 (0.085)	0.008 (0.018)	0.004 (0.094)	−0.084 (0.245)
Observations	49	49	49	49
R^2	0.321	0.321	0.351	0.289
Adjusted R^2	0.224	0.224	0.258	0.188

Note. $^{\dagger}p < .10$. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

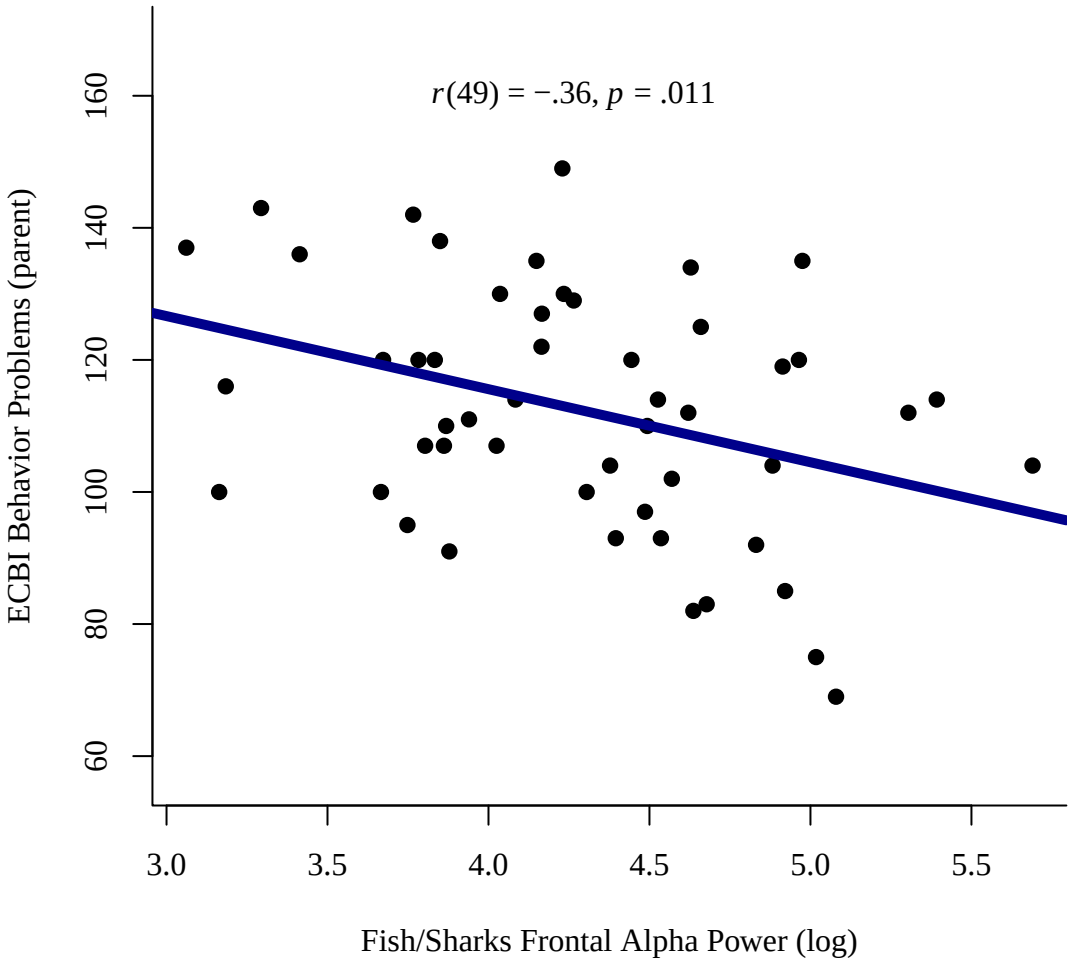

FIGURE 3.38: Association between frontal alpha power during the Fish/Sharks task and parent-reported intensity of behavior problems on the ECBI.

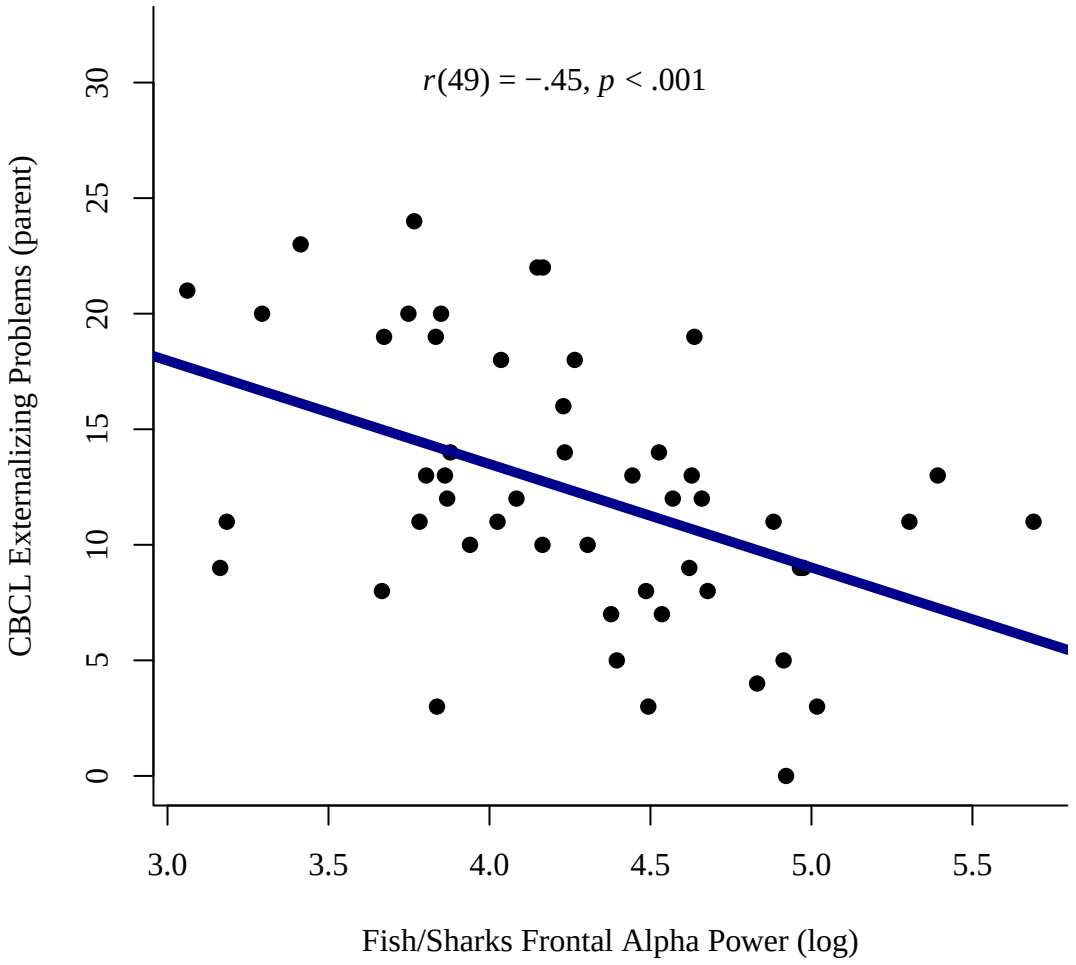

FIGURE 3.39: Association between frontal alpha power during the Fish/Sharks task and parent-reported CBCL Externalizing Problems.

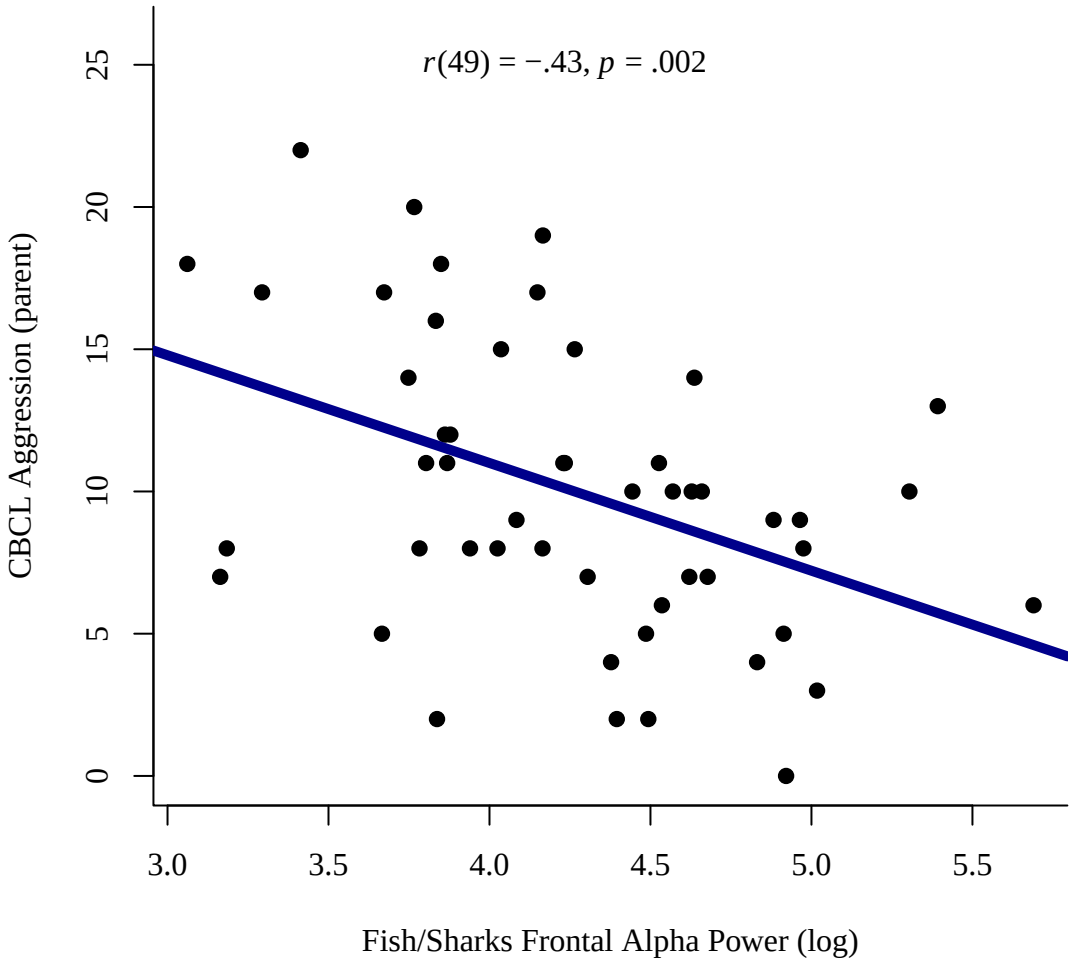

FIGURE 3.40: Association between frontal alpha power during the Fish/Sharks task and parent-reported CBCL Aggression.

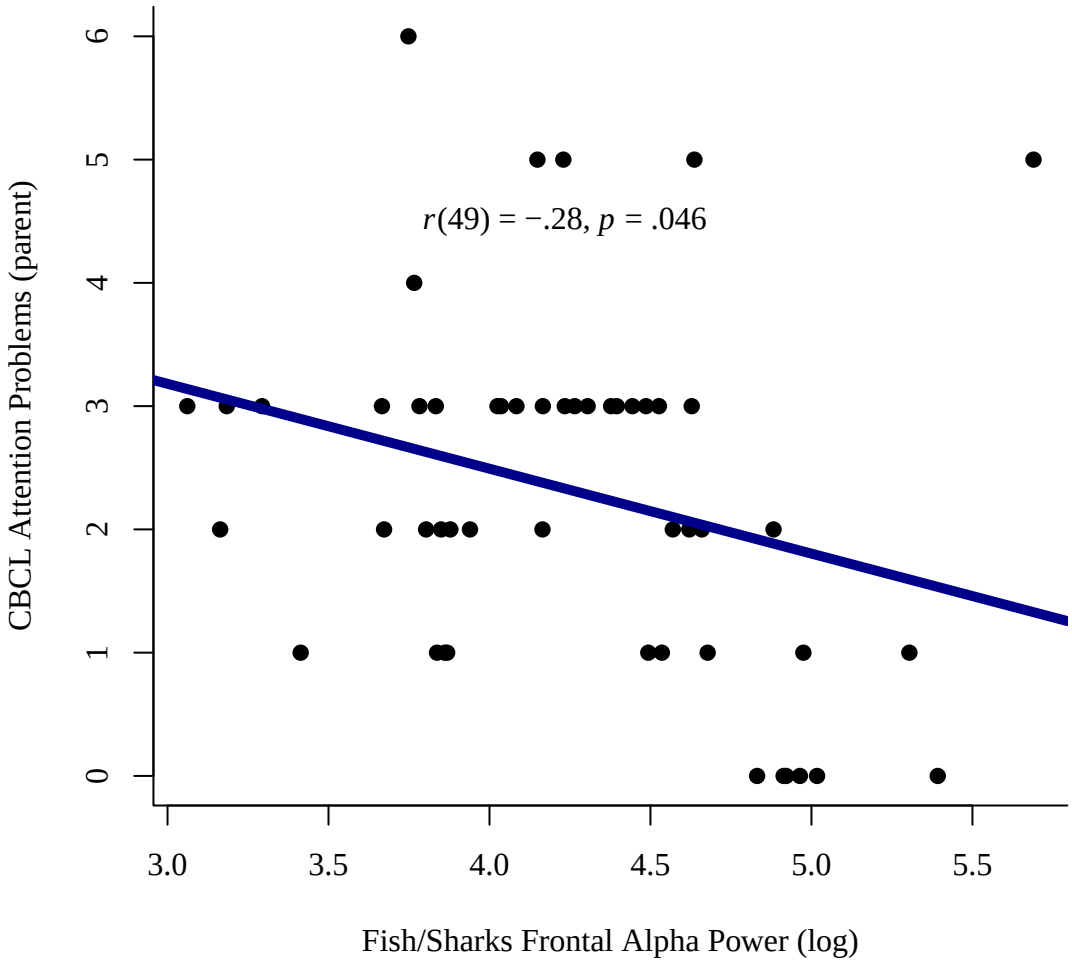

FIGURE 3.41: Association between frontal alpha power during the Fish/Sharks task and parent-reported CBCL Attention Problems.

TABLE 3.23: Study 2: Clustered Regression Examining Association of Left Frontal Asymmetry with Aggression and Externalizing Problems.

	Dependent variable:		
	ECBI Behavior Problems (Parent)	CBCL Externalizing Problems (Secondary Caregiver)	CBCL Aggression (Secondary Caregiver)
Intercept	122.415*** (24.643)	−10.527 (8.493)	−5.733 (7.308)
Left Frontal Asymmetry (Oddball)	13.681* (5.383)		
Left Frontal Asymmetry (Fish/Sharks)		3.446 (2.980)	3.229† (1.897)
Sex	−0.376 (5.452)	0.028 (2.151)	0.924 (1.543)
Age	5.057 (4.575)	2.115 (1.468)	0.653 (1.053)
Number of Bad Channels	−1.288	0.261 (0.457)	0.074 (0.302)
Number of Target Trials Kept	−0.814 (0.763)		
Number of No-Go Trials Kept		−0.156 (0.288)	−0.177 (0.239)
Behavioral Percent Correct on No-Go Trials		0.084 (0.079)	0.091 (0.063)
Observations	76	19	19
R^2	0.119	0.292	0.286
Adjusted R^2	0.056	-0.062	-0.071

Note. $^{\dagger}p < .10$. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

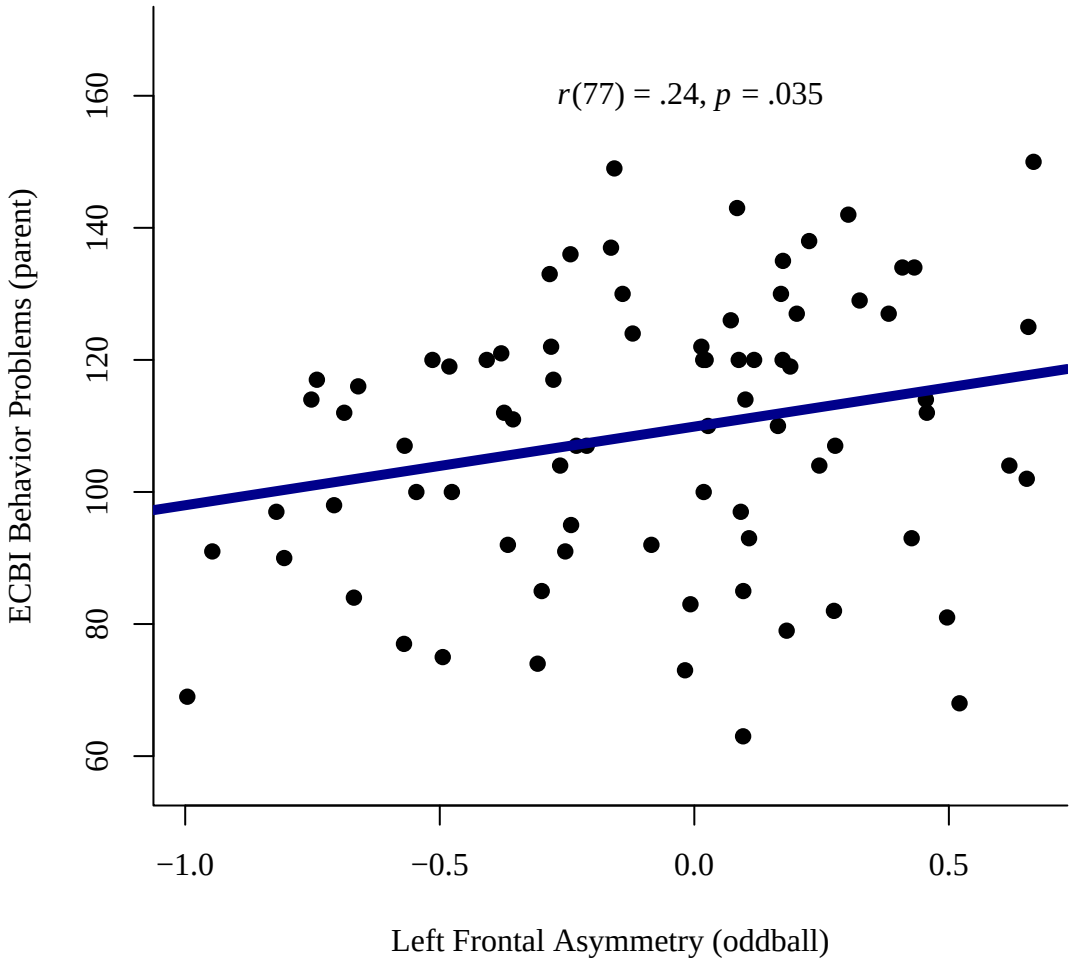

FIGURE 3.42: Association between left frontal asymmetry during the oddball task and parent-reported intensity of behavior problems on the ECBI.

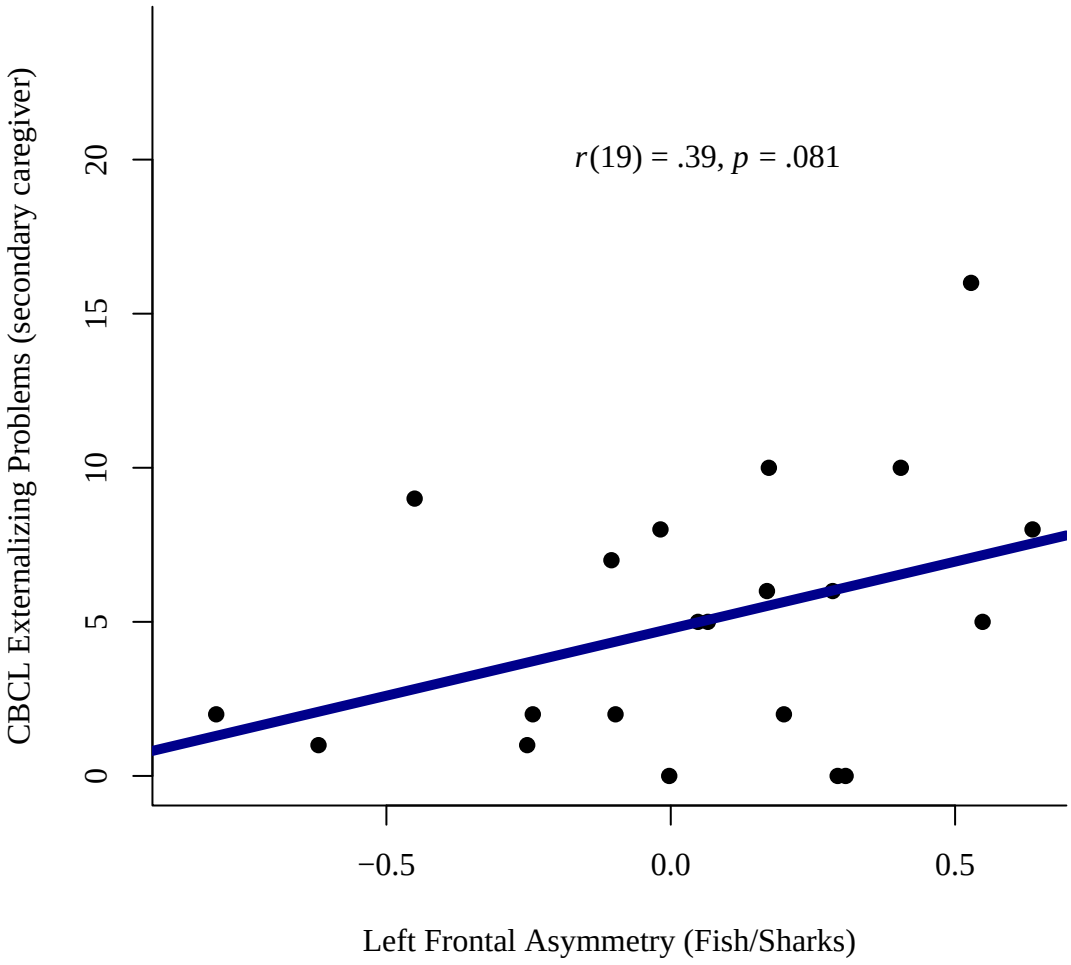

FIGURE 3.43: Association between left frontal asymmetry during the Fish/Sharks task and secondary caregiver-reported CBCL Externalizing Problems.

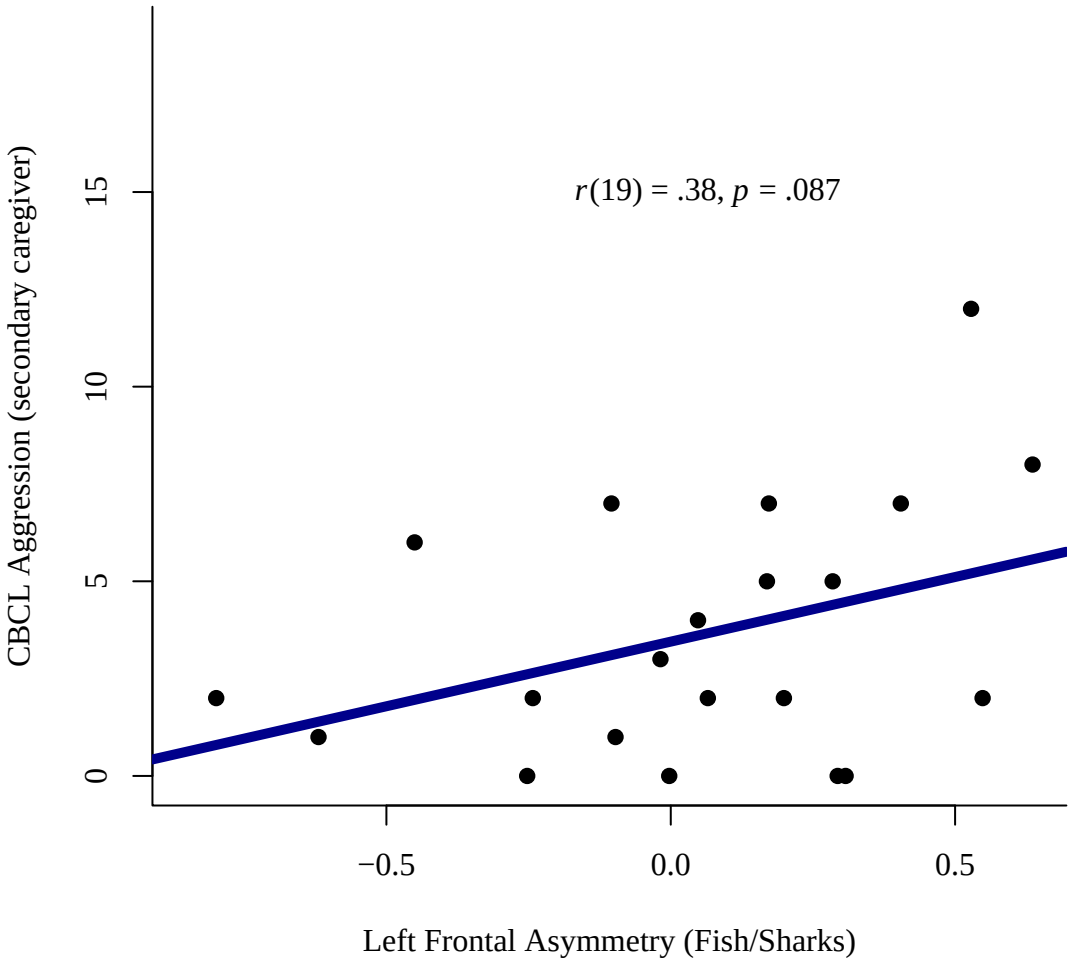

FIGURE 3.44: Association between left frontal asymmetry during the Fish/Sharks task and secondary caregiver-reported CBCL Aggression.

Less frontal alpha power in the Fish/Sharks task remained associated with later aggression ($p = .006$), externalizing problems ($p = .006$), and ECBI behavior problems ($p = .014$) when examining Spearman's rho and when accounting for the nesting of longitudinal data, controlling for covariates, and controlling for prior levels of behavior problems (see Table 3.24).

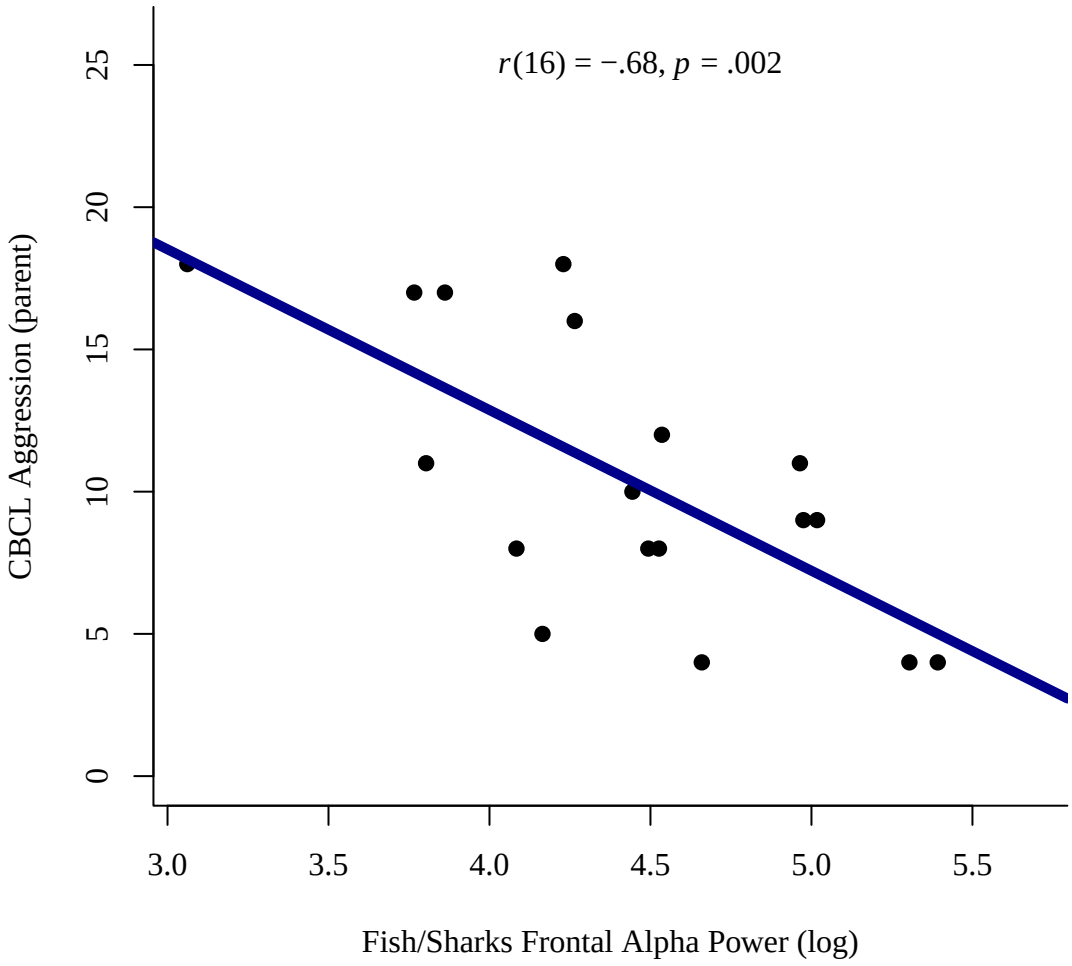

FIGURE 3.45: Association between frontal alpha power during the Fish/Sharks task and later parent-reported CBCL Aggression.

TABLE 3.24: Study 2: Pearson Correlations of Children's EEG and Time-Frequency Components with their Externalizing Problems (Lagged).

	Oddball			Fish/Sharks		
	Frontal Power	Frontal Asymmetry	Frontal TF	Frontal Power	Frontal Asymmetry	Frontal TF
CBCL EXT Parent	−.12	−.21	−.16	−.65**	.19	.22
CBCL EXT Secondary	.51	−.40	−.07	−.18	−.40	.84
CBCL AGG Parent	−.09	−.21	−.12	−.68**	.14	.20
CBCL AGG Secondary	.62	−.41	−.08	−.03	−.54	.94†
CBCL ATT Parent	−.16	−.13	−.23	−.31	.36	.25
CBCL ATT Secondary	.02	−.24	−.02	−.46	−.05	.52
ECBI Intensity Parent	−.10	.10	−.17	−.62**	−.16	.03

Note. "TF" = time-frequency activity corresponding to timing of P3a (oddball) or N2 (Fish/Sharks), with values in decibels. "EXT" = externalizing problems, "AGG" = aggression, "ATT" = attention problems. Power values were log-transformed. Frontal power and asymmetry in alpha frequency range. Frontal time-frequency in theta frequency range. Frontal asymmetry reflects right frontal alpha power − left frontal alpha power (i.e., higher values reflect left frontal asymmetry in alpha frequency range). Correlations are two-tailed.

TABLE 3.25: Study 2: Clustered Regression Examining Association of Frontal Alpha Power (Fish/Sharks) with Later Aggression and Externalizing Problems.

	Dependent variable:		
	Lagged CBCL Aggression (parent)	Lagged CBCL Externalizing Problems (parent)	Lagged ECBI Behavior Problems (parent)
Intercept	55.027** (17.133)	52.675** (16.347)	132.259*** (35.092)
Frontal Alpha Power (Fish/Sharks)	−6.865*** (1.592)	−6.555*** (1.828)	−11.696*** (2.707)
Sex	−0.586 (1.655)	0.030 (1.876)	7.839 (4.947)
Age	−9.910** (3.803)	−12.165** (4.479)	−38.364*** (10.631)
Number of Bad Channels	0.091 (0.226)	0.304 (0.260)	−1.207* (0.486)
Number of No-Go Trials Kept	0.160 (0.496)	0.029 (0.518)	0.708 (0.735)
Behavioral Percent on No-Go Trials	0.090 (0.126)	0.139 (0.131)	0.928** (0.317)
CBCL Aggression	0.290 (0.328)		
CBCL Externalizing Problems		0.544 (0.355)	
ECBI Behavior Problems			0.526*** (0.100)
Observations	17	17	16
R^2	0.665	0.668	0.846
Adjusted R^2	0.405	0.410	0.711

Note. $^{\dagger}p < .10$. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

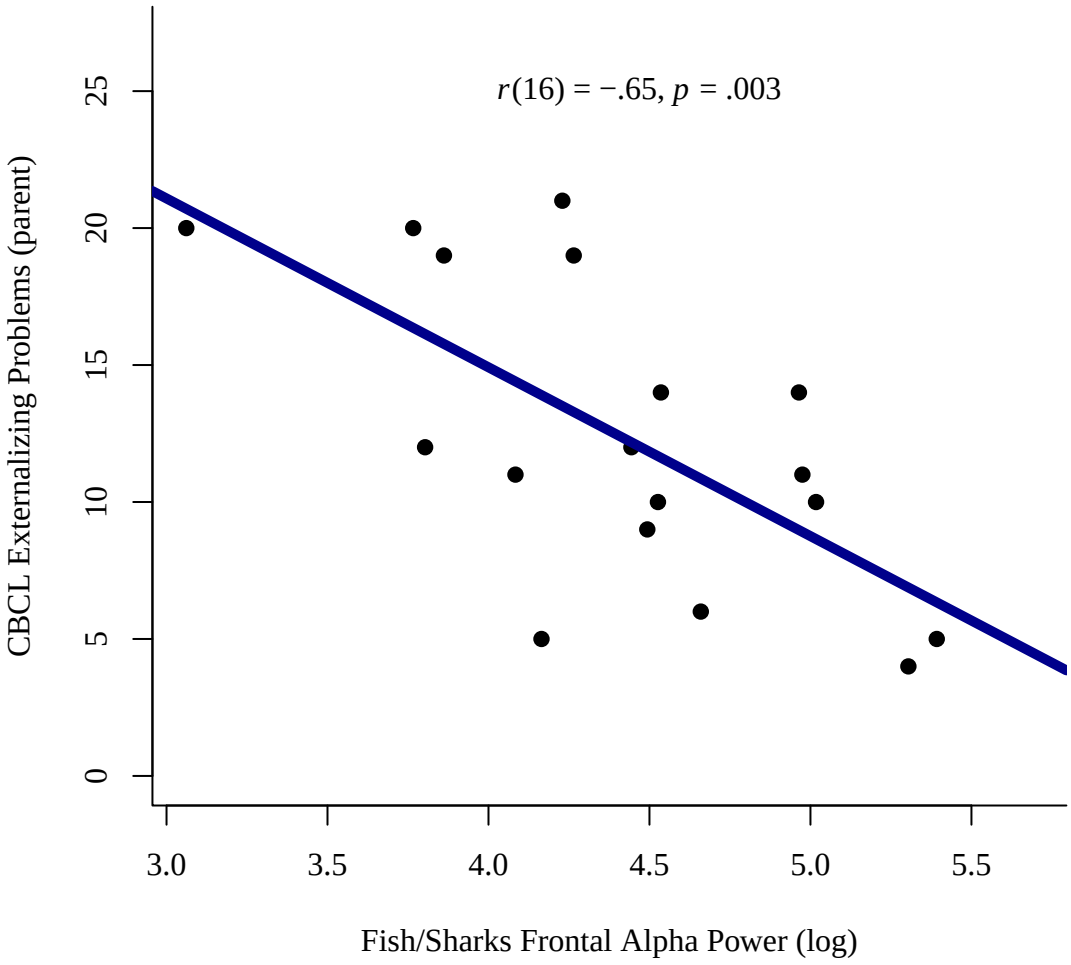

FIGURE 3.46: Association between frontal alpha power during the Fish/Sharks task and later parent-reported CBCL Externalizing Problems.

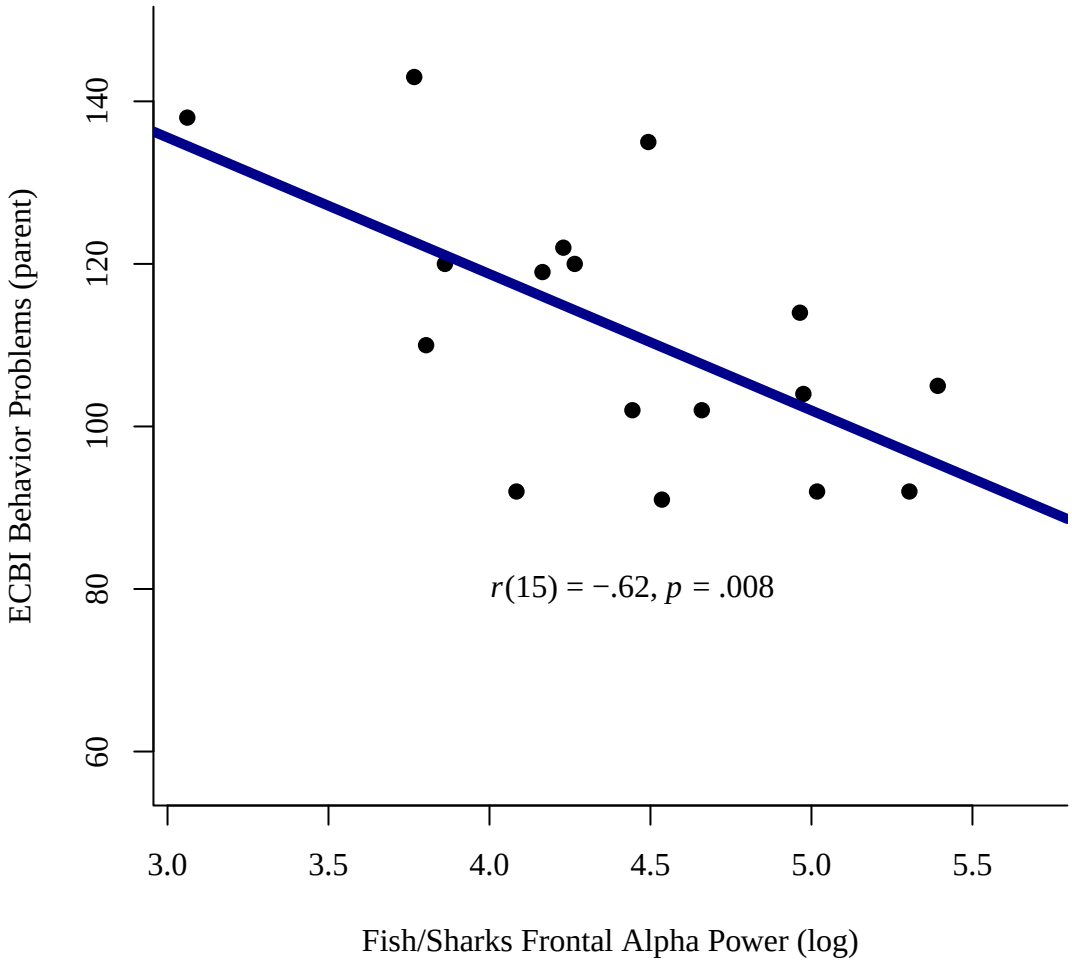

FIGURE 3.47: Association between frontal alpha power during the Fish/Sharks task and later parent-reported intensity of behavior problems on the ECBI.

3.2.2.0.3 Time-Frequency Neurophysiology and Externalizing Problems.

Pearson correlations of children's time-frequency values and asymmetry scores with their concurrent externalizing problems are in Table 3.20. Consistent with hypotheses, less P3a-related frontal theta activity was associated with more parent-reported externalizing problems ($r[77] = -.23$, $p = .045$, see Figure 3.48) and aggression ($r[77] = -.24$, $p = .031$, see Figure 3.49). Less P3a-related frontal theta activity remained associated with later externalizing problems ($p = .033$) and aggression ($p = .024$) when examining Spearman's rho and when accounting for the nesting of longitudinal data and controlling for covariates (see Table 3.26).

TABLE 3.26: Study 2: Clustered Regression Examining Association of P3a-Related Frontal Theta Activity with Externalizing Problems and Aggression.

	Dependent variable:	
	CBCL Aggression (parent)	CBCL Externalizing Problems (parent)
Intercept	$13.370^{\dagger}$ (7.677)	$10.454^{\dagger}$ (6.094)
P3a-Related Frontal Theta Activity	-0.357^{*} (0.150)	-0.318^{*} (0.135)
Sex	−2.319 (1.570)	−1.653 (1.328)
Age	1.217 (1.516)	1.055 (1.318)
Number of Bad Channels	−0.225 (0.281)	−0.223 (0.219)
Number of Target Trials Kept	−0.108 (0.193)	−0.061 (0.158)
Observations	76	76
R^2	0.118	0.118
Adjusted R^2	0.055	0.055

Note. $^{\dagger}p < .10$. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

Pearson correlations of children's time-frequency values and asymmetry scores with their later externalizing problems are in Table 3.24. Only one (marginally significant) association was observed, and it was inconsistent with hypotheses. More N2-related frontal theta activity was associated with later secondary-caregiver aggression.

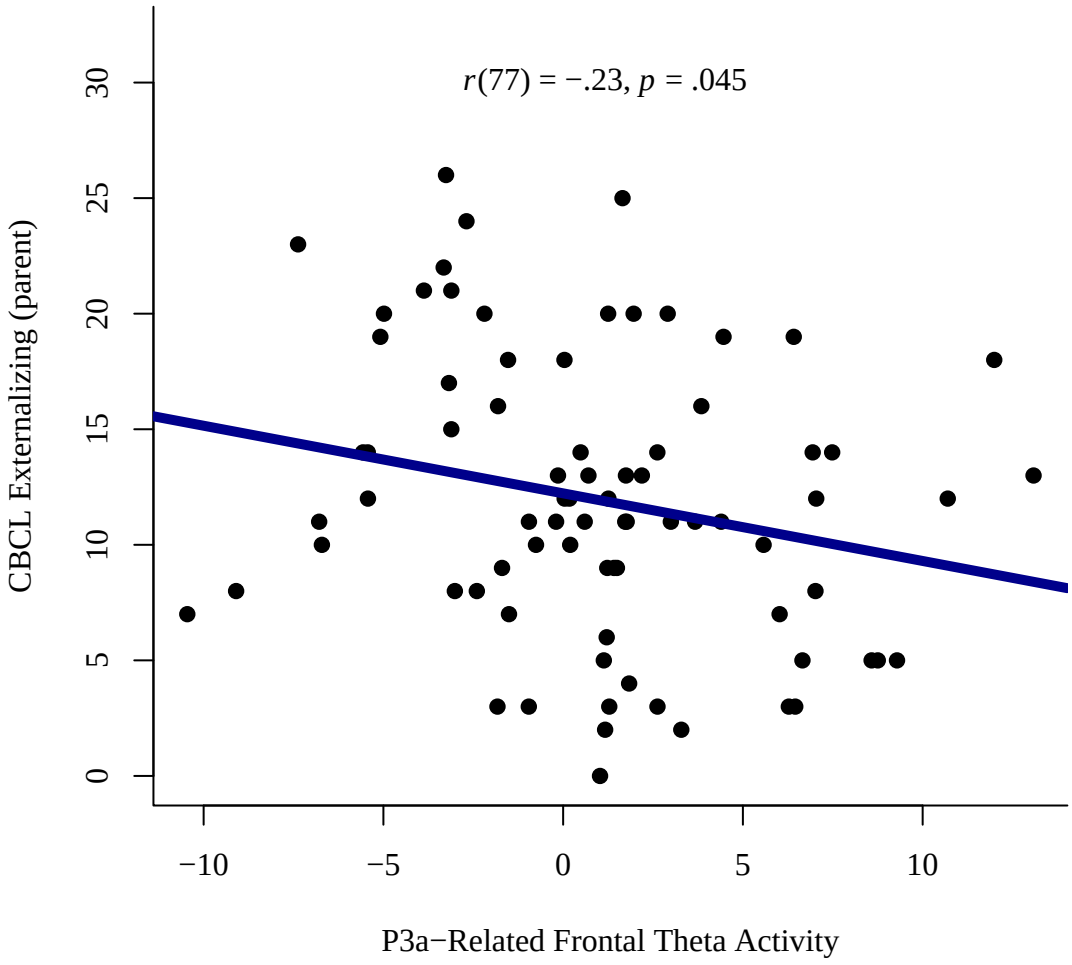

FIGURE 3.48: Association between P3a-related frontal theta activity and parent-reported CBCL Externalizing.

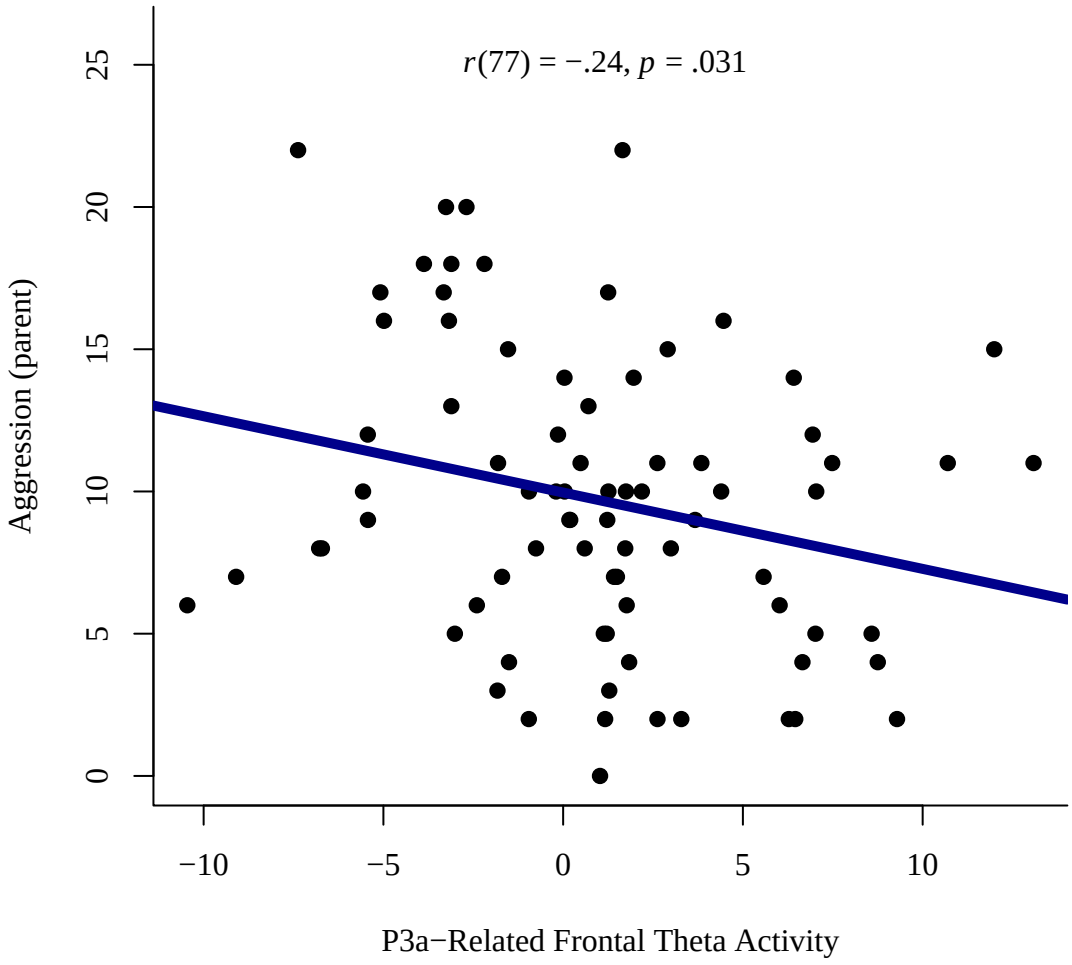

FIGURE 3.49: Association between P3a-related frontal theta activity and parent-reported CBCL Aggression.

3.2.3 Association Between Self-Regulation and Externalizing Problems

For more accurate estimates of the association between self-regulation and behavior problems, we used the full sample of 336 families who were part of the larger study (i.e., not just those who were recruited for the EEG procedures). Pearson correlations between children's self-regulation and their concurrent behavior problems are in Table 2.23. Pearson correlations between children's self-regulation and their later behavior problems are in Table 3.27. Concurrent associations between self-regulation and behavior problems were described in Study 1. Because of the greater rate of longitudinal follow-up in Study 2 compared to Study 1, we examined longitudinal mediation models based on which self-regulation variables were associated with later behavior problems.

All statistically significant associations were consistent with hypotheses. Poorer performance on Bird/Alligator was associated with later parent-reported ECBI behavior problems, CBCL externalizing and attention problems, and secondary caregiver-reported externalizing, aggression, and attention problems. Poorer performance on Shape Stroop was associated with later secondary caregiver-reported aggression. Poorer performance on Fish/Sharks was associated with later parent-reported attention problems.

3.2.4 Mediation

Three self-regulation variables were associated with later behavior problems: Bird/Alligator, Shape Stroop, and Fish/Sharks. Based on associations of neurophysiological variables with later self-regulation on these tasks, we tested two possible mediational processes with Shape

TABLE 3.27: All Studies: Pearson Correlations of Children's Self-Regulation with Ratings of Their Externalizing Problems (Lagged).

	CBCL EXT	CBCL EXT (Sec)	CBCL AGG	CBCL AGG (Sec)	CBCL ATT	CBCL ATT (Sec)	ECBI
Bird/Alligator	$-.11^{\dagger}$	$-.21^{*}$	$-.09$	$-.18^{\dagger}$	$-.12^{\dagger}$	$-.21^{*}$	$-.14^{*}$
Shape Stroop	$-.04$	$-.12$	$-.03$	$-.15^{\dagger}$	$-.09$	.01	.01
Grass/Snow	$-.01$	$-.18$	$-.03$	$-.17$	.07	$-.11$	$-.08$
Token Sort	.09	$-.12$	.09	$-.12$	.05	$-.09$	.06
Sustained Play Attention	.05	$-.08$	.04	$-.07$	.08	$-.05$	.04
Fish/Sharks	$-.02$	.06	.08	.02	$-.40^{*}$	.12	$-.01$

Note. $^{\dagger}p < .10$. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$. "EXT" = Externalizing Problems. "AGG" = Aggression. "ATT" = Attention Problems. "Sec" = secondary caregiver-reported. Correlations are two-tailed. *N*s range from 77 to 282 (except for correlations with performance on the Fish/Sharks ERP task, which was administered only to a subset of the whole sample, with *N*s ranging from 11 to 31).

Stroop and Fish/Sharks (no neurophysiological variables predicted later performance on Bird/Alligator):

1. Longer no-go N2 latencies → poorer inhibitory control in the Fish/Sharks task → more attention problems
2. Less N2-related frontal theta activity → poorer inhibitory control in Shape Stroop → more aggression

Longer no-go N2 latencies predicted poorer subsequent performance on Fish/Sharks, and poorer performance on the Fish/Sharks task was associated with later parent-reported attention problems, so we examined this as a possible mediational process. However, there were insufficient cases with longitudinal Fish/Sharks data at 30 and 36 months (5) to examine whether the association between N2 latencies at 30 months and behavior problems at 42 months was mediated by performance on the same task at 36 months (when controlling for prior levels). Less N2-related frontal theta activity was associated with poorer subsequent performance on Shape Stroop, and poorer performance on Shape Stroop was associated with later secondary caregiver-reported aggression, so we examined this as a possible mediational process. Again, however, there were insufficient cases with longitudinal reports of aggression by secondary caregivers at 36 and 42 months (11) to examine whether the association between N2-related frontal theta activity at 30 months and aggression at 42 months was mediated by Shape Stroop performance at 36 months (when controlling for prior levels). Thus, because of the modest amount of longitudinal data, we were unable to fit full-longitudinal mediation models to test these developmental processes.

On the other hand, we were able to fit half-longitudinal mediation models (Cole & Maxwell, 2003) by collapsing T1–T2 (30–36 month) associations with T2–T3 (36–42 month) associations, which tests a mediation model with only two time points and assumes stationarity of relations across time. In half-longitudinal models, Fish/Sharks performance did not mediate the association between N2 latencies and later attention problems (CI: −0.14, 0.09), and Shape Stroop performance did not mediate the association between N2-related frontal theta activity and later aggression (CI: −0.13, 0.14). In sum, we found no evidence that self-regulation mediated the associations between neurophysiological variables and later behavior problems, for the measures examined.

3.2.5 Sensitivity and Specificity

To investigate the potential clinical utility of neurophysiological variables as predictors of later behavior problems, we examined ROC curves to determine the tradeoff between sensitivity and specificity of prediction. Only one neurophysiological variable predicted later behavior problems in ways that were consistent with hypotheses: less frontal alpha power in the Fish/Sharks task predicted later parent-reported externalizing, aggression, and attention problems, controlling for plausible confounds and prior levels of behavior problems (see Table 3.25). We examined the sensitivity and specificity of frontal alpha power to classify concurrent and predict later high levels of externalizing problems.

Out of 51 cases with data for both frontal alpha power in the Fish/Sharks task and concurrent parent-reported externalizing problems, 8 (16%) were considered to have high levels of externalizing problems. An ROC curve of frontal alpha power in classifying concurrent externalizing problems is in Figure 3.50. Frontal alpha power had an area under the curve

(AUC) of .85 in classifying concurrent high levels of externalizing problems. The optimal cutpoint depends on the assessment goal (Treat & Viken, 2012). The sum of sensitivity and specificity was greatest at a cutpoint of 4.17 (i.e., children were classified with high levels of externalizing problems with log-transformed frontal alpha power values below 4.17), with a sensitivity of 1.00 and a specificity of .63. This cutpoint meets our goals for an optimal cutpoint on a screening measure because it places greater emphasis on identifying at-risk children (i.e., sensitivity) than on avoiding false positives (i.e., specificity).

Out of 18 cases with data for both frontal alpha power in the Fish/Sharks task and later parent-reported externalizing problems, 3 (17%) were considered to have high levels of externalizing problems. Frontal alpha power had an AUC of .91 in predicting later high levels of externalizing problems. The sum of sensitivity and specificity was greatest at a cutpoint of 4.23 (i.e., children were classified with high levels of externalizing problems with log-transformed frontal alpha power values below 4.23), with a sensitivity of 1.00 and a specificity of .73.

3.2.6 Effect Sizes

In examining effect sizes, we focused on replicating patterns of effects. Longer P3a latencies were associated with sustained attention ($|r| \approx .6$) and externalizing, aggression, and attention problems ($|r| \approx .3$) with medium-to-large effect sizes. Quadratic associations of N2 amplitudes with self-regulation ($|r| \approx .2$ to .3) and attention problems ($|r| \approx .6$) showed medium-to-large effect sizes. Left frontal asymmetry was associated with poorer self-regulation, externalizing problems, and aggression with small-to-medium effect sizes

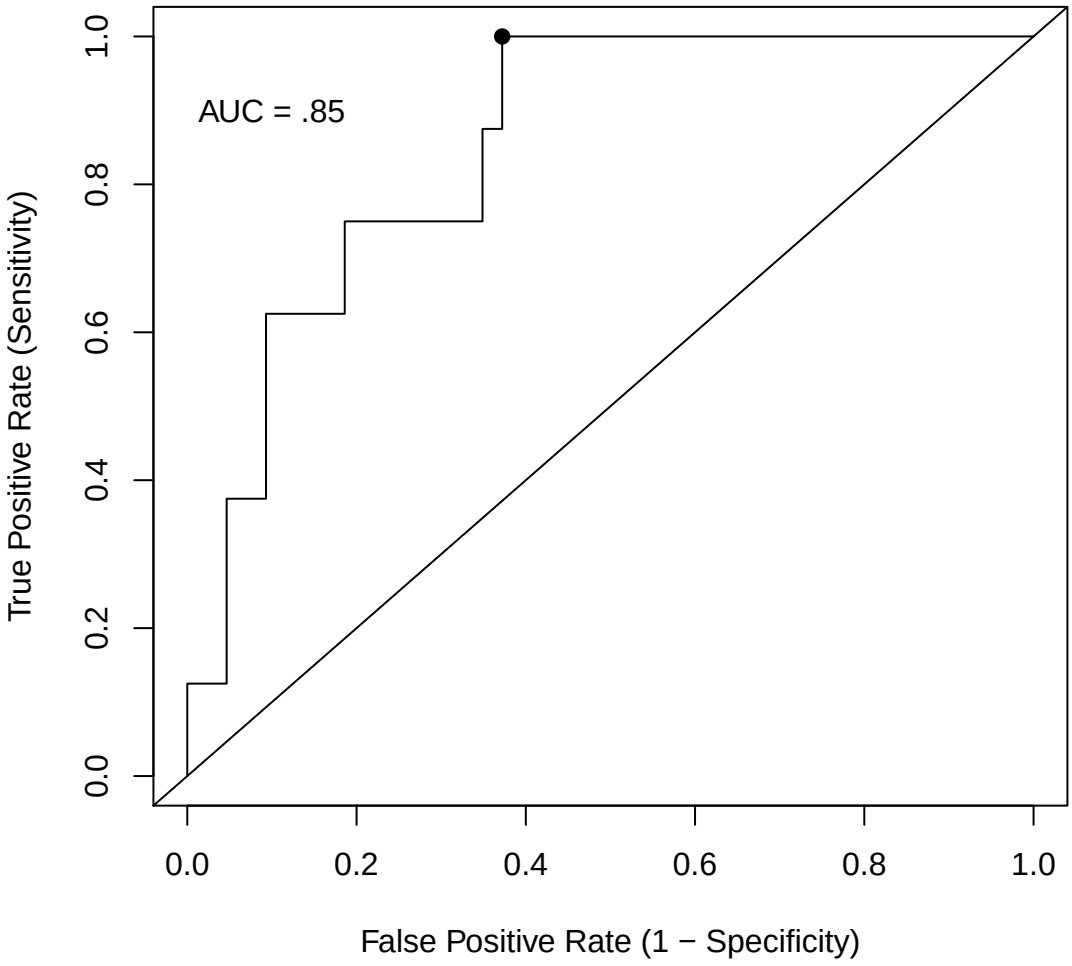

FIGURE 3.50: Receiver operating characteristics (ROC) curve of frontal alpha power in the classification of high levels of externalizing problems. The dot represents the optimal cutpoint for log-transformed frontal alpha power values (4.17), defined as the cutpoint with the highest sum of sensitivity (1.00) and specificity (.63).

($|r| \approx .2$ to .4). Less frontal alpha power in the oddball task was associated with externalizing problems and aggression with small effect sizes ($|r| \approx .2$). Less frontal alpha power in the Fish/Sharks task was associated with concurrent externalizing, aggression, and attention problems with medium effect sizes ($|r| \approx .3$ to .4). Less frontal alpha power in the Fish/Sharks task predicted later levels of externalizing, aggression, and attention problems with large effect sizes ($|r| \approx .6$ to .7). Associations between self-regulation and later externalizing problems were based on a larger sample, so we had greater power to detect effects. Most observed associations between self-regulation and later externalizing problems had small effect sizes ($|r| \approx .1$ to .2).

3.3 Discussion

Study 2 examined concurrent and longitudinal associations between neural functioning, self-regulation, and externalizing behavior. There was some evidence that neural functioning predicted self-regulation. Larger N2 amplitude difference scores and shorter target P3a latencies predicted better subsequent performance on Token Sort. Shorter no-go N2 latencies predicted better subsequent performance on Fish/Sharks. Nonlinear (quadratic) associations of no-go N2 amplitudes with Shape Stroop and Token Sort were also observed, with very large or very small N2 amplitudes associated with poorer self-regulation. Left frontal asymmetry in the oddball task was concurrently associated with worse performance on Sustained Play Attention, and left frontal asymmetry in the Fish/Sharks task was concurrently associated with poorer performance on Grass/Snow. More N2-related frontal theta activity predicted better subsequent performance on Shape Stroop.

Some associations between neural functioning and self-regulation were inconsistent with hypotheses. Smaller P3a amplitude difference scores predicted better subsequent performance on Grass/Snow. Smaller no-go N2 amplitudes were associated with better concurrent performance on Bird/Alligator. Smaller N2 amplitude differences scores were associated with better concurrent performance on Bird/Alligator and Fish/Sharks, and with better subsequent performance on Token Sort. Longer no-go N2 latencies were associated with better concurrent performance on Shape Stroop, and with better subsequent performance on Grass/Snow. Less frontal alpha power in both the oddball and Fish/Sharks tasks was associated with better concurrent and later performance on Token Sort. More N2-related frontal theta activity was associated with better concurrent performance on Token Sort.

There was also evidence of neural functioning predicting externalizing problems. Longer target P3a latencies were concurrently associated with externalizing, aggression, and attention problems. A nonlinear (quadratic) association between no-go N2 amplitudes and attention problems was observed, with very large or very small N2 amplitudes associated with more attention problems. Less frontal alpha power in both the oddball and Fish/Sharks tasks was associated with concurrent levels of externalizing problems and aggression (less frontal alpha power in the Fish/Sharks task was also associated with attention problems). Less frontal alpha power in the Fish/Sharks task also predicted later externalizing problems and aggression. Left frontal asymmetry in both the oddball and Fish/Sharks tasks was concurrently associated with externalizing problems, and left frontal asymmetry in the Fish/Sharks task was also associated with aggression. P3a-related frontal theta activity was concurrently associated with externalizing problems and aggression.

Some associations between neural functioning and externalizing problems were inconsistent with hypotheses. Larger no-go N2 amplitudes were associated with more concurrent externalizing and attention problems and with later attention problems (but the association with concurrent attention problems was better accounted for by a quadratic than linear association). Shorter no-go N2 latencies predicted later externalizing problems and aggression, and N2-related frontal theta activity predicted later aggression, but this was based on very few cases with later reports by secondary caregivers.

Regarding the quadratic associations of no-go N2 amplitudes with self-regulation and attention problems, findings suggest that there may be an optimal range of no-go N2 amplitudes. Children whose no-go N2 amplitudes were relatively large (< -10 μV) or small (> 0 μV) tended to show poorer self-regulation and more attention problems, but it is unclear how reliable the effect was because it may have been driven by outliers.

Only one neurophysiological variable predicted later externalizing problems in ways that were consistent with hypotheses: less frontal alpha power in the Fish/Sharks task predicted later parent-reported externalizing, aggression, and attention problems, controlling for plausible confounds and prior levels of behavior problems. Less frontal alpha power showed moderate accuracy in classifying concurrent levels and predicting later levels of externalizing problems. Regarding longitudinal mediational models, there was no evidence that self-regulation mediated the associations between neural functioning and later externalizing problems.

In summary, there was no evidence in support of Hypothesis 1 that externalizing problems would be associated with smaller amplitudes of the no-go N2 (although both smaller

and larger no-go N2 amplitudes were associated with more attention problems). Rather, *larger* no-go N2 amplitudes tended to be associated with poorer self-regulation and more externalizing problems. There was also no evidence for Hypothesis 2 that externalizing problems would be associated with longer latencies of the no-go N2. There was no evidence for Hypothesis 3 that externalizing problems would be associated with smaller amplitudes of the oddball P3a. On the other hand, there was some support for Hypothesis 4 that externalizing problems would be associated with longer latencies of the oddball P3a. There was some support for Hypothesis 5 that externalizing problems would be associated with left frontal asymmetry in the alpha frequency band. There was fairly robust support for Hypothesis 6 that externalizing problems would be associated with less frontal alpha power, and both concurrent and lagged associations were observed. There was some support for Hypothesis 7 that externalizing problems would be associated with less P3a-related frontal theta activity, but no support for hypotheses of associations with N2-related frontal theta activity. Although shorter P3a latencies were associated with better sustained attention, there was no support for Hypotheses 8 and 9 that the no-go N2 and oddball P3a would show differential associations with disinhibition (N2) and sustained attention (P3a). Finally, there was no support for Hypothesis 10 that self-regulation would mediate the association between neural functioning and later externalizing problems.

Chapter 4

General Discussion

4.1 Description of Studies

The present studies examined the relation between neurophysiology, self-regulation, and externalizing behavior problems in very young children. Study 1 examined the concurrent associations of candidate neurophysiological markers with self-regulation and externalizing behavior problems among 27 children who were 2 1/2 to 3 1/2 years old. Eight of the 27 children had multiple EEG assessments, resulting in 35 cases. Study 2 examined the concurrent and lagged associations of candidate neurophysiological markers with self-regulation and externalizing behavior problems among 64 children (87 cases) who were also 2 1/2 to 3 1/2 years old. It is important to examine the incremental validity of neural measures over behavioral measures when predicting externalizing problems to demonstrate the added value of neural measures (Youngstrom & Reyes, 2015). In Study 2, we examined whether neural functioning predicted later externalizing problems controlling for prior levels of externalizing problems. To our knowledge, the present report is the first to examine neural functioning in a go/no-go task in children younger than 4 years of age (for examples of studies using go/no-go tasks with 4-year-olds, see Chevalier et al., 2014; Lahat et al., 2010; Lewis, Todd,

& Honsberger, 2007; Moreno, Bialystok, Barac, Schellenberg, Cepeda, & Chau, 2011; Todd, Lewis, Meusel, & Zelazo, 2008), and the first to examine neural functioning in an active oddball task in 2–3-year-old children (apart from Hoyniak et al., in press, whose data were a subset of those in Study 1 of the present report).

Although both studies included a go/no-go (N2) and oddball (P3) ERP task, there were key differences between both studies. The go/no-go task in Study 1 was a Bird/Alligator task with an equal ratio of go and no-go trials. The go/no-go task in Study 2 was a Fish/Sharks task with an unequal ratio of go and no-go trials (75% go, 25% no-go). The oddball task in Study 1 was an *active* oddball paradigm with child-friendly sounds (duck quack, cat meow) designed to elicit the P3b ERP. The oddball task in Study 2 was a *passive* two-tone oddball paradigm without a behavioral response designed to elicit the P3a ERP. In Study 1, we included incorrect trials in the subject average waveforms (for a rationale, see Section 2.1.4), whereas in Study 2, we excluded trials in which an incorrect behavioral response was made.

4.2 Summary of Findings

In interpreting the findings, we give more weight to findings from Study 2 than Study 1 because of the larger sample in Study 2, but we give the greatest weight to those findings that were replicated across studies and measures.

4.2.1 Did We Identify a No-Go N2 and Oddball P3?

In both studies, a frontocentral negativity was observed in the go/no-go task and a posterior positivity was observed in the oddball task. The question is whether these ERP components reflect the N2/P3 components commonly observed in older children. In both studies, the no-go N2 was larger in amplitude than the go N2, suggesting that it may reflect inhibitory processing. Moreover, the frontocentral electrode cluster we identified in Study 2 where the N2 was most maximal was similar to the left-lateralized frontal electrode cluster identified by Chevalier, Kelsey, Wiebe, and Espy (2014) in 5-year-olds using the same Fish/Sharks task. However, the target P3a/b components were not larger than the frequent P3a/b. This calls into question whether the posterior positivity identified in the oddball tasks of the present studies actually reflects the same attentional processes characterized by the P3 ERP component in older children. We cannot rule out the possibility that the posterior positive component is a P3-like component that changes in meaning with development (reflecting a developing cognitive process related to attentional processing). On the other hand, it does appear that we were able to identify the no-go N2 in very young children. The meaning of the no-go N2 likely changes with development, as well, given the heterotypic continuity of inhibitory control (i.e., inhibitory control changes in form with development; Petersen, Hoyniak, McQuillan, et al., under review). Nevertheless, to our knowledge, the present report is the first investigation of go/no-go and active oddball ERP tasks in 2–3-year-olds (but for an example of a passive oddball task in 2-year-olds, see Niemitalo-Haapola et al., 2013), so we present our cognitive interpretations of these components with caution.

In terms of descriptive statistics, the peak latencies of the N2 and P3 ERPs were somewhat different in both studies. The N2 peak latency was 572 ms in Study 1 and was 392 ms in Study 2. The P3b peak latency was 716 ms in Study 1, and the P3a peak latency was 442 ms. We would expect the P3b to have a longer latency than the P3a, but the magnitude of this difference was surprising. Likewise, we were surprised by the magnitude of difference between latencies of the N2 in Study 1 and Study 2. There were a number of key methodological differences between Study 1 and Study 2 (described above), so it is unclear whether latency differences across studies related to any of these or other methodological differences. For example, it is possible that incorrect trials may result in longer latencies than correct trials, which may have partially accounted for the latency differences between the two studies. In terms of amplitude, the no-go N2 had a mean amplitude of -9.91 μV in Study 1 and -7.18 μV in Study 2. The target P3a had a mean amplitude of 8.11 μV in Study 1, and the target P3b had a mean amplitude of 6.76 μV in Study 2.[1]

4.2.2 Reliability, Validity, and Change

We also considered the cross-time continuity, convergent validity, discriminant validity, and developmental change of neurophysiological markers. The only markers that demonstrated cross-time continuity were no-go N2 latencies (Study 1: $r = .80$, although this was based on only 6 children) and frontal alpha power in the Fish/Sharks task (Study 2: $r = .63$). Frontal alpha power (Study 1: $r = .67$, Study 2: $r = .54$) and frontal asymmetry scores (Study

[1]The grand average waveforms depicted in Figures 2.6, 2.11, 3.7, and 3.12 represent the mean waveform from those electrodes with a 0.4 or greater factor loading onto the PCA component reflecting the relevant N2/P3 component; electrodes were averaged with equal, unit weighting. The ERP amplitudes in Tables 2.1 and 3.1 were calculated from PCA. In the PCA, all electrodes contribute to the estimation of amplitudes to the extent that they reflect the underlying N2/P3 component (based on factor loadings), thus more heavily weighting those electrodes that are driving the signal. This accounts for the larger amplitudes in Table 2.1 than in Figures 2.6 and 2.11 (Study 1) and in Table 3.1 than Figures 3.7 and 3.12 (Study 2).

1: $r = .40$, Study 2: $r = .25$) showed convergent validity across tasks, whereas N2/P3-related frontal theta activity did not (Study 1: $r = .06$, Study 2: $r = -.04$). We would expect less correspondence between N2- and P3-related frontal theta activity because the time-frequency values were calculated using different time windows and presumably reflect different cognitive processes specific to the N2/P3 component of interest. Frontal alpha power and frontal asymmetry also showed discriminant validity with different measures from the same task (see Tables 2.4 and 3.4). Both studies showed that frontal alpha power had stronger cross-time continuity and convergent validity than did frontal asymmetry. This is consistent with previous findings that frontal alpha power is more reliable than frontal asymmetry in childhood (Vuga et al., 2008). Regarding developmental changes, frontal alpha power decreased with age in the Fish/Sharks task; otherwise, there were no detectable, reliable developmental changes from 2 1/2 to 3 1/2 years of age for any of the other neurophysiological markers examined.

4.2.3 Hypotheses

We tested ten hypotheses (see Section 1.4.3). There was no evidence in support of Hypothesis 1 that externalizing problems would be associated with smaller amplitudes of the no-go N2 (although both smaller *and* larger no-go N2 amplitudes were associated with more attention problems in Study 2). Rather, *larger* no-go N2 amplitudes tended to be associated with poorer self-regulation and more externalizing problems (Study 2). There was also no evidence for Hypothesis 2 that externalizing problems would be associated with longer latencies of the no-go N2. On the other hand, there was some support for Hypothesis 3 that externalizing problems would be associated with smaller amplitudes of the oddball P3

(Study 1). There was support for Hypothesis 4 that externalizing problems would be associated with longer latencies of the oddball P3 (Studies 1 and 2). There was some support for Hypothesis 5 that externalizing problems would be associated with left frontal asymmetry in the alpha frequency band (Study 2). There was fairly robust support for Hypothesis 6 that externalizing problems would be associated with less frontal alpha power, and both concurrent (Studies 1 and 2) and lagged (Study 2) associations were observed. There was some support for Hypothesis 7 that externalizing problems would be associated with less P3a-related frontal theta activity (Study 2), but no support for hypotheses of associations with N2-related frontal theta activity. There was no support for Hypotheses 8 and 9 that the no-go N2 and oddball P3b would show differential associations with disinhibition measures (N2) and sustained attention measures (P3b). Finally, there was a little cross-sectional (Study 1) but no longitudinal support for Hypothesis 10 that self-regulation would mediate the association between neural functioning and later externalizing problems.

Across the two studies, 12 (5%) of the 228 tested associations between neurophysiological functioning and self-regulation were consistent with hypotheses ($p < .1$, two-tailed), about the same as would be expected simply by chance. Fifteen (7%) of the 228 tested associations between neurophysiological functioning and self-regulation were inconsistent with hypotheses, which is slightly higher than would be expected by chance. As a result, there was not a strong pattern of associations between neurophysiological functioning and self-regulation. Twenty-eight (11%) of the 252 tested associations between neurophysiological functioning and behavior problems were consistent with hypotheses, more than twice the number that would be expected by chance. Twelve (5%) of the 252 tested associations

between neurophysiological functioning and behavior problems were inconsistent with hypotheses, about the same as would be expected by chance. Thus, there was a stronger pattern of associations between neurophysiological functioning and behavior problems that was consistent with hypotheses.

4.2.4 Association of ERPs with Self-Regulation and Externalizing Problems

The no-go N2 is thought to reflect processes of inhibitory control (response inhibition) or conflict monitoring (Nakata, Inui, Wasaka, Tamura, et al., 2006; Nieuwenhuis et al., 2003; Rueda, Posner, et al., 2004; J. L. Smith et al., 2008; van Veen & Carter, 2002a; Yeung et al., 2004), and the oddball P3 is thought to reflect attentional processing (Key et al., 2005). N2 and P3 ERPs showed some associations with self-regulation and behavior problems in ways that were consistent with hypotheses. Smaller P3b amplitudes were associated with poorer self-regulation (inhibitory control) and externalizing behavior problems. Longer P3a and P3b latencies were associated with externalizing behavior problems and aggression. Longer P3a latencies were also associated with inattentive/hyperactive problems (i.e., CBCL Attention Problems). These findings suggest that (a) less recruitment of attentional processes (i.e., smaller P3 amplitudes) in a context requiring sustained attention and (b) less efficient attentional processing (longer P3 latencies) might relate to poorer self-regulation and behavioral adjustment. Only one association between the P3a and later self-regulation was observed. Longer P3a latencies were associated with poorer sustained attention 6 months later, but did not remain associated with later sustained attention when controlling for plausible confounds and prior levels of sustained attention. Thus, although there was considerable evidence of concurrent associations of P3 amplitudes/latencies with self-regulation

and behavior problems, the oddball P3 did not predict changes over time in self-regulation or behavior problems.

Examining the N2, smaller N2 amplitude difference scores (smaller difference between no-go N2 amplitude and go N2 amplitude) were associated with poorer inhibitory control (Study 1). On the other hand, larger (more negative) N2 amplitudes were associated with poorer self-regulation and behavior problems (Study 2), but a nonlinear association with N2 amplitudes appeared to account for some of these effects. There was evidence of a quadratic association of N2 amplitudes with inhibitory control (Shape Stroop), sustained attention (Token Sort), and inattentive/hyperactive problems (secondary caregiver reports on the CBCL Attention Problems subscale). Evidence suggested that there was an optimal range of N2 amplitudes from approximately -10 to 0 μV.[2] Children with larger or smaller N2 amplitudes tended to show poorer self-regulation and inattentive/hyperactive problems. This might suggest a new model for understanding how inhibitory processing might relate to self-regulation and behavioral adjustment. For instance, given that the no-go N2 has neural sources in the PFC and ACC (Jonkman, Sniedt, et al., 2007; Lamm et al., 2006; Stieben et al., 2007), children with excessively small no-go N2 amplitudes may have a hypoactive PFC and ACC that leads to impaired ability to inhibit prepotent responses. On the other hand, children with excessively large no-go N2 amplitudes may recruit excess PFC and ACC activity that is unnecessary for inhibiting responses. N2 amplitudes normatively decrease with development in childhood (Broyd et al., 2005; Ciesielski et al., 2004; E. P.

[2]Note that 0 μV does not reflect the absence of neural activity because it is a measure of relative not absolute activity—i.e., neural activity at a given electrode is relative to other electrodes and to the 200 ms prestimulus baseline (and neural activity that is detectable on the scalp reflects the firing of millions of neurons).

Davis et al., 2003; Hämmerer, Li, Müller, et al., 2010; Henderson, 2010; Johnstone, Dimoska, et al., 2007; Johnstone, Pleffer, et al., 2005; Jonkman, 2006; Jonkman, Lansbergen, et al., 2003; Jonkman, Sniedt, et al., 2007; Lamm et al., 2006; Lewis, Lamm, et al., 2006), so excessively large N2 amplitudes may reflect developmentally immature neural inhibitory processing. Speculatively, excessively large N2 amplitudes may correspond to findings of a developmental lag in brain maturity in the PFC (indexed by cortical thickness) in ADHD (Shaw, Eckstrand, et al., 2007). If children recruit too many inhibitory resources, their processing may be less efficient/automatic and may be more effortful, taking away important processing resources from other tasks. Thus, excessively large N2 amplitudes may reflect inefficiency in neural inhibitory processing that leads to compensation on simple behavioral tasks requiring inhibition of a prepotent response. When there are numerous competing demands and complex/extraneous stimuli, however, these children with inefficient inhibitory processing may be unable to inhibit prepotent demands. We present the behavioral consequences of excessively small or large N2 amplitudes as speculative directions for future research to consider. Nevertheless, our findings are supported by findings in rats that attention deficits can result from both hypo- and hyper-activation of the medial prefrontal cortex (Pezze et al., 2014).

The finding of a quadratic association of N2 amplitudes with self-regulation and attention problems might account for the inconsistent direction of previous findings. Some have found that self-regulation and externalizing-related problems are associated with smaller N2 amplitudes (Albrecht et al., 2005; Berger et al., 2013; Broyd et al., 2005; Cragg et al., 2009; Dimoska, Johnstone, Barry, & Clarke, 2003; Falkenstein et al., 1999; Grabell, 2014; Jetha et al., 2010; Johnstone, Barry, & Clarke, 2007; Johnstone, Barry, Markovska, et al., 2009;

Lahat et al., 2010; Overtoom, Verbaten, et al., 1998; Pliszka, Liotti, et al., 2000; Schmajuk et al., 2006; van Boxtel et al., 2001; Wiersema & Roeyers, 2009; Wild-Wall et al., 2009), whereas others have found associations with larger N2 amplitudes (Dimoska, Johnstone, & Barry, 2006; Espinet et al., 2012; Kok et al., 2004; Ramautar et al., 2004, 2006; Senderecka, Grabowska, Szewczyk, et al., 2012; J. L. Smith et al., 2004; Woltering et al., 2011), and yet others have found no association (e.g., Spronk et al., 2008). As can be seen in Figure 1.1, if N2 amplitudes demonstrate a quadratic association with externalizing problems, one might find a positive, negative, or even null association depending on the particular distribution/range of the N2 amplitudes in which the correlation is examined. Moreover, the quadratic model might also account for the seeming paradox that although studies typically find smaller N2 amplitudes in a range of self-regulation and behavior problems, N2 amplitudes normatively decrease over time in childhood. In other words, findings from previous studies suggest that behavior problems may be characterized by more "developmentally mature" N2 amplitudes! If the association is quadratic, however, it could explain why, even though the optimal N2 amplitudes decrease with development, small amplitudes can be associated with behavior problems when they are *too* small (i.e., hypoactive rather than efficient/focalized PFC/ACC processing during inhibitory demands). Nevertheless, the quadratic association may have owed, in part, to outliers, so we hope future studies will examine the possibility that N2 amplitudes have a nonlinear association with self-regulation and behavior problems.

N2 latencies were not reliably associated with self-regulation or behavior problems in ways that were consistent with hypotheses. Only one association between the N2 and later

self-regulation was observed that was consistent with hypotheses. Children with longer no-go N2 latencies showed poorer inhibitory control in the same Fish/Sharks task 6 months later, even when controlling for plausible confounds and prior levels of inhibitory control. Findings suggest that slower, less efficient inhibitory control processing may relate to poorer development of inhibitory control, but this association was not replicated across other measures of inhibitory control and was not observed when examining concurrent associations, so we present it with caution. In sum, when examining longitudinal prediction of neurophysiological ERP measures in relation to later self-regulation and behavior problems, there were markedly fewer associations.

4.2.5 Association of EEG with Self-Regulation and Externalizing Problems

We examined two EEG measures: frontal alpha power (6–9 Hz frequency band) and frontal hemispheric asymmetry. Frontal alpha power is thought to be inversely correlated with cortical activity (Fox, 1994), and higher levels of frontal alpha power may reflect the inhibition of task-irrelevant neural activity (Klimesch, 2012; Klimesch, Sauseng, & Hanslmayr, 2007). Thus, less frontal alpha power in the context of a task requiring sustained attention (active oddball) or inhibitory control (go/no-go) might reflect the recruitment of excess neural processes for sustained attention or inhibitory control, and might reflect less neural efficiency. Consistent with hypotheses and prior findings in relation to inhibitory control (Wolfe & Bell, 2004), we observed less frontal alpha power in relation to behavior problems in both studies. We observed concurrent associations between behavior problems and frontal alpha power, as measured during all four tasks. However, we only observed predictive associations between frontal alpha power and later behavior problems when considering frontal alpha

power measured during the go/no-go task and not the passive oddball task. This suggests that frontal alpha power may be most relevant to the development of externalizing problems when considering neural processing in a context requiring inhibitory control or sustained attention (unlike a passive oddball task). Associations between frontal alpha power and later behavior problems remained when controlling for plausible confounds and when controlling for prior levels of behavior problems, suggesting that children with less frontal alpha power showed relative increases in behavior problems over time compared to children with more frontal alpha power.[3] The associations were specific to alpha (and not theta) power, but were not specific to frontal electrodes—similar associations were observed at posterior electrodes. Less frontal alpha power during a go/no-go task was also associated with poorer inhibitory control in the same task (Study 1). Interestingly, however, less frontal alpha power in both the passive oddball and go/no-go tasks was associated with *better* sustained attention, both concurrently and predictively (Study 2). We hope that future studies examine the meaning of frontal alpha power in very early childhood during inhibitory and attentional processing.

We also examined frontal hemispheric asymmetry, hypothesizing that left frontal asymmetry would be associated with more externalizing behavior problems. Left frontal asymmetry is thought to reflect behavioral approach tendencies, whereas right frontal asymmetry is thought to reflect behavioral withdrawal tendencies (Coan & Allen, 2004). We observed a trend of left frontal asymmetry as measured during a go/no-go task in relation to secondary caregiver-reported externalizing problems and aggression (Study 2). We did not observe associations with frontal hemispheric asymmetry as measured during the oddball

[3]Predicting later behavior problems while controlling for prior levels allows us to predict relative rank-order change in behavior problems, not absolute increases or decreases in level.

task, or when examined in relation to parent-reported behavior problems or lagged behavior problems, or in Study 1. The inconsistent associations between left frontal asymmetry and externalizing problems may owe to the types of tasks used in the present studies. Studies have typically examined frontal asymmetry in the context of a stressful task designed to elicit emotional reactivity and individual differences in approach/withdrawal. Our tasks were designed to tap processes of inhibitory control and attentional processing rather than emotion regulation. Nevertheless, our general lack of associations between left frontal asymmetry and externalizing problems is consistent with findings of a meta-analysis in children that there was no association between left frontal asymmetry and externalizing problems (although the meta-analysis did find an association between *right* frontal asymmetry and internalizing problems; Peltola et al., 2014).

4.2.6 Association of Time-Frequency Neurophysiology with Self-Regulation and Externalizing Problems

We also examined time-frequency neurophysiology—N2- and P3-related frontal theta activity—in relation to self-regulation and behavior problems. Based on prior findings of less P3-related frontal theta activity in adults with alcoholism (Jones et al., 2006; Porjesz, Rangaswamy, et al., 2005), we hypothesized that less N2- and P3-related frontal theta activity would be associated with poorer self-regulation and behavior problems. Consistent with hypotheses, less P3a-related frontal theta activity was associated with more parent-reported externalizing problems and aggression (Study 2). However, findings with N2-related frontal theta activity were more mixed. Consistent with hypotheses, less N2-related frontal theta activity predicted worse inhibitory control performance 6 months later on Shape Stroop.

On the other hand, less N2-related frontal theta activity was concurrently associated with *better* self-regulation in both studies and with more parent-reported behavior problems in Study 1. Thus, findings with P3a-related frontal theta activity were consistent with findings from prior studies in adults, but associations with N2-related frontal theta were mixed.

4.2.7 Association Between Self-Regulation and Externalizing Problems

Unexpectedly, self-regulation variables showed modest (i.e., small effect sizes; see Section 4.2.10) and inconsistent associations with externalizing problems. As might be expected, poorer inhibitory control on Bird/Alligator was associated with more inattentive/hyperactive problems concurrently, and more externalizing, aggression, and inattentive/hyperactive problems 6 months later. Poorer sustained attention in Token Sort was concurrently associated with more externalizing and inattentive/hyperactive problems. Poorer inhibitory control in Shape Stroop was associated with more aggression 6 months later, and poorer inhibitory control in Fish/Sharks was associated with more inattentive/hyperactive problems 6 months later. In summary, there was some but limited evidence that poorer self-regulation was associated with externalizing problems.

4.2.8 Did Self-Regulation Mediate the Association Between Neural Functioning and Externalizing Problems?

We tested whether self-regulation mediates the association between neural functioning and externalizing problems to determine whether self-regulation represents an underlying, intermediate phenotype of externalizing problems. We found possible evidence of one cross-sectional mediational process: P3b amplitudes had an indirect effect on secondary caregiver-reported externalizing problems via inhibitory control as measured by Bird/Alligator (Study 1). Smaller P3b amplitudes were associated with poorer inhibitory control and more externalizing problems, and poorer inhibitory control partially mediated the association between P3b amplitudes and externalizing problems. The mediation model included a small sample size and there are difficult assumptions of cross-sectional mediation models, however, so we present this finding with caution. We found no evidence of longitudinal mediation.

In summary, there were modest and inconsistent associations between behavioral measures of self-regulation and behavior problems and little evidence that self-regulation mediated the association between neurophysiology and behavior problems. Thus, the evidence suggests that, although self-regulation deficits may be related to behavior problems, behavioral self-regulation may not be as core to behavior problems as originally thought (insofar as self-regulation and behavior problems were measured in the present studies). Findings also suggest that the self-regulation tasks we examined would not serve as good proxy variables for neural measures when relating these neural measures to behavioral adjustment. As a result, it appears that the neural measures provide incremental validity above the behavioral measures in associations with behavior problems. Nevertheless, self-regulation

encompasses a broad construct and the measures in the current studies do not fully capture its breadth, so we hesitate to generalize our findings to other behavioral measures relevant to self-regulation.

4.2.9 Sensitivity and Specificity

Because of the importance of examining sensitivity and specificity of measures for establishing their clinical utility (Youngstrom & Reyes, 2015), we examined ROC curves of neural functioning predicting externalizing problems. Only one neurophysiological variable predicted later behavior problems in ways that were consistent with hypotheses: less frontal alpha power in the Fish/Sharks task predicted later parent-reported externalizing, aggression, and attention problems, controlling for plausible confounds and prior levels of behavior problems. We examined the sensitivity and specificity of frontal alpha power to classify concurrent and predict later externalizing problems. ROC curves demonstrated moderate accuracy for classifying concurrent (AUC = .85) and predicting later (AUC = .91) high levels of externalizing problems (above the normed 80th percentile). We defined the optimal cutpoint as the cutpoint with the greatest sum of sensitivity and specificity. This resulted in optimal cutpoints of 4.17 and 4.23 for classifying concurrent and predicting later externalizing problems, respectively. Sensitivity was 1.0 at these cutpoints for classifying concurrent and predicting later externalizing problems, respectively. Specificity was .63 and .73 at these cutpoints for classifying concurrent and predicting later externalizing problems, respectively. These cutpoints meet our goal for an optimal cutpoint on a screening measure because they place greater emphasis on identifying at-risk children (i.e., sensitivity)

than on avoiding false positives (i.e., specificity). Findings suggest that children with log-transformed frontal alpha power values of less than approximately 4.17–4.23 in the context of no-go trials of a go/no-go task may be at risk for developing externalizing problems. Future research should attempt to replicate and extend our findings with a larger sample.

4.2.10 Effect Sizes

We also considered the effect size of associations among neural functioning, self-regulation, and externalizing problems. Smaller oddball target P3b amplitudes and smaller P3b amplitude difference scores were associated with poorer self-regulation in the Bird/Alligator task ($|r| \approx .4$) and more secondary caregiver-rated externalizing problems ($|r| \approx .8$) with medium-to-large effect sizes, but the latter association was based on a small sample with secondary caregiver reports. Longer oddball P3a latencies were associated with later sustained attention in the Token Sort task ($|r| \approx .6$) and concurrent externalizing, aggression, and attention problems ($|r| \approx .3$) with medium-to-large effect sizes. Quadratic associations of N2 amplitudes with self-regulation ($|r| \approx .2$ to .3) and attention problems ($|r| \approx .6$) showed medium-to-large effect sizes. Left frontal asymmetry was associated with poorer self-regulation (Grass/Snow and Sustained Attention), externalizing problems, and aggression with small-to-medium effect sizes ($|r| \approx .2$ to .4). Less frontal alpha power during a passive oddball task was associated with parent-reported externalizing problems and aggression with small effect sizes ($|r| \approx .2$). Less frontal alpha power during an active oddball task and go/no-go task were associated with poorer self-regulation ($|r| \approx .3$), concurrent parent-reported externalizing problems ($|r| \approx .3$ to .6), and later parent-reported externalizing

problems ($|r| \approx .6$ to .7) with medium-to-large effect sizes. Associations of self-regulation with concurrent and later externalizing problems had small effect sizes ($|r| \approx .1$ to .2).

These associations were generally robust to outliers and covariates (sex, age, the number of bad channels, the number of trials kept, and the behavioral performance on the task) and some were even replicated across studies/samples, suggesting that these patterns of associations might reflect meaningful neural mechanisms in the development of self-regulation and externalizing behavior. Nevertheless, there could be other important third variables that explain the association between the neural markers examined and externalizing behavior, so we plan to continue following the children longitudinally to gain a better sense of the developmental process and the brain–behavior relations in early childhood.

Interestingly, we observed stronger associations of frontal alpha power with concurrent and later externalizing problems than we did with any other neurophysiological measure, and we observed this association across a range of measures of behavior problems, across measurements of frontal alpha power from four different tasks, and across two samples, providing further confidence in our findings. The finding of strongest associations with frontal alpha power may reflect that it was the most reliable neurophysiological measure we examined in terms of cross-time continuity, and it also showed strong convergent validity with respect to the same measure across tasks and discriminant validity with respect to other neurophysiological measures from the same task. Whereas ERPs reflect a snapshot of a particular cognitive process and may need a sizable number of trials for an adequate signal-to-noise ratio to capture this cognitive process, EEG measures such as alpha power may reflect a more broad-band measure of neural functioning that might demonstrate more rank-order stability.

4.3 Limitations

The present studies had several limitations. First, the samples were community samples, so it remains to be seen whether the findings would generalize to a sample of children with externalizing disorders. We used community samples because we were interested in the underlying processes in the development of externalizing problems and wanted a sample representative of a full range of risk to characterize variations around normality in self-regulation and externalizing behavior. Nevertheless, a number of children (Study 1: 4, Study 2: 10) were above the 80th percentile on externalizing problems relative to a norm-referenced sample, suggesting that the processes we identified may operate similarly for some children with severe disruptive behavior. However, the sample was fairly high in SES and educational attainment, with little ethnic diversity (predominantly Caucasian). Future studies should examine neural mechanisms of psychopathology in more nationally-representative samples.

Another limitation may have been a modest power to detect effects because of a small sample (Study 1) and a relatively low signal-to-noise ratio (both studies). In both studies, the signal-to-noise ratio of the EEG waveforms may be somewhat low compared to that from studies with adults. Toddlers have a limited attentional capacity, so we had to keep the tasks fairly brief (approximately 6 minutes) with fewer trials than most ERP studies with adults. Fewer trials administered, in combination with more motor movement during these trials and incorrect responses to a number of trials, led to fewer trials retained than in most ERP studies with adults. Thus, it is likely that the signal-to-noise ratio was somewhat lower in our study than in many studies with adults. However, this may be counteracted, in part,

by findings that the no-go N2 amplitude is greatest in early childhood and decreases with age from childhood to adulthood (Broyd et al., 2005; Ciesielski et al., 2004; E. P. Davis et al., 2003; Hämmerer, Li, Müller, et al., 2010; Henderson, 2010; Johnstone, Dimoska, et al., 2007; Johnstone, Pleffer, et al., 2005; Jonkman, 2006; Jonkman, Lansbergen, et al., 2003; Lewis, Lamm, et al., 2006; Rueda, Fan, et al., 2004). Because we had specific hypotheses and modest power to detect effects, we did not adjust for multiple testing, so findings should be interpreted in light of the fact that our test-wise (not family-wise) Type I error rate was .05 (i.e., we would expect 5% of associations to be identified simply by chance). Thus, we placed the greatest emphasis on *patterns* of associations and replications of findings across studies and measures. Nevertheless, the present studies are among the first to examine neural functioning in relation to self-regulation and externalizing problems in toddlerhood, a key developmental era for the development of positive adjustment, so we feel the theoretically-guided exploratory approach was useful.

Also of note, we calculated theta and alpha power using fixed bands across children rather than determining individual-specific bands, which would have been preferable (Klimesch, 1999). We calculated fixed frequency bands because we did not have baseline data on all children to determine each child's theta–alpha transition frequency (i.e., theta power increases from baseline to task and alpha power decreases from baseline to task). Nevertheless, we calculated theta and alpha power using somewhat narrow frequency bands (encompassing a range not wider than 3–4 Hz), so we would likely not be combining much from outside (i.e., non-theta/alpha) frequency bands. Moreover, even if calculations included some outside frequency bands for some children, doing so would have worked against our hypotheses because theta and alpha power have partially inverse effects (Schmid, Tirsch, & Reitmeir,

1997), so combining them would have made it *less* likely to observe the hypothesized effects.

Additionally, because we examined neural functioning in very young children, we had to adapt our procedures to accommodate the population of interest. In Study 1, we included incorrect trials in the analysis (i.e., participants' average waveforms were calculated across trials whether or not a behavioral response was made; for a more detailed rationale, see Section 2.1.4). By contrast, in Study 2, we included only correct trials in the EEG/ERP analyses because Study 2 used task paradigms and procedures that were more developmentally appropriate for young children, including a more child-friendly go/no-go task with performance feedback. A limitation of including only correct trials in Study 2 was that it resulted in a higher rate of missingness than would have been observed had we included incorrect trials, particularly among the youngest children. Future studies should examine the number of usable trials necessary per condition to have an adequate signal-to-noise ratio to observe the ERP components of interest in young children. It would also be useful for studies to determine the degree to which findings differ when including incorrect trials, as was done in Hoyniak, Petersen, McQuillan, Staples, and Bates (in press). Excluding incorrect trials, although conforming to the traditions in the ERP literature, resulted in more missingness than would be acceptable for clinical assessment. For neurophysiological measures to be clinically useful, they should have a lower missingness rate than was observed in our studies (40% missingness in Study 1, 33% in Study 2). The neurophysiological literature should determine the best way to accommodate young children and clinical populations to retain as much useful information as possible for a greater percentage of cases.

4.4 Strengths

The present studies also had key strengths. First, the studies used a longitudinal design with repeated measures of neurophysiological and behavioral data. The longitudinal design allowed us to examine the cross-time rank-order stability of neurophysiological and behavioral measures, and to examine the co-development of brain functioning and behavioral adjustment in a very important developmental era. The cross-lagged analyses of Study 2 allowed us to examine whether brain functioning predicted later change in behavioral adjustment. Second, we used a multitrait-multimethod approach to the measurement of self-regulation and externalizing behavior, with multiple lab tasks, questionnaires, raters, and indices of neural functioning. Third, we applied an RDoC approach to examine neural functioning in relation to more specific behavioral indices thought to reflect underlying phenotypes of externalizing behavior, including self-regulation phenotypes of inhibitory control and sustained attention. This allowed us to better understand the meaning of the neural markers we examined, including the no-go N2 and oddball P3a/b. Fourth, we used two mostly independent samples that allowed us to examine the replicability of our findings. The replication of some findings provides greater confidence in these results.

Chapter 5

Conclusion and Future Directions

5.1 Future Directions

Findings that less efficient neural processing in inhibitory control or sustained attention may lead to self-regulation and externalizing problems in young children lead to several possible future directions. We plan to follow the children from this study longitudinally into school age with repeated measures of neural functioning and adjustment. This will allow us to examine whether the early neural markers predict later behavior problems during an important developmental transition when there are more demands placed on children and more opportunities for misbehavior. We also plan to examine neural functioning in relation to school readiness, another important dimension of behavioral adjustment. Doing so will allow us to better understand the early mechanisms in the development of positive behavioral and school adjustment, and may allow earlier, more targeted identification of at-risk children. Future studies should also develop more successful intervention programs for children with or at risk for deficits in inhibitory control, sustained attention, or externalizing behavior. To the extent that research can identify specific mechanisms in the development

of externalizing behavior (e.g., smaller no-go N2 amplitudes resulting in inhibitory control deficits), interventions might be designed to target these specific mechanisms.

5.2 So We Might Be Able To Identify At-Risk Children Early On...Then What?

Understanding the brain mechanisms underlying the development of externalizing behavior problems allows not only better early identification of children at risk for developing externalizing problems, but also may allow the development of better, more specific treatments that target these mechanisms (Insel, 2014). Better interventions may include more specific pharmacological treatments that have greater efficacy and fewer side effects. Alternatively, although evidence is speculative at this point, future interventions may involve cognitive training (especially if targeting multiple neuropsychological domains; Cortese et al., 2015) or neurofeedback (Gevensleben et al., 2009; but previous studies have questioned the clinical utility of neurofeedback; Vollebregt, van Dongen-Boomsma, Buitelaar, and Slaats-Willemse, 2014). This "brain training," for instance, may involve training to increase the capacity or efficiency for neural response inhibition via the N2 or frontal alpha power. As our understanding of psychopathology and behavior increasingly focuses on brain circuits, however, interventions may shift toward manipulating neural circuits through deep brain stimulation or optogenetics. Deep brain stimulation, the stimulation of particular brain regions with electrodes implanted in the brain, has shown promising efficacy in treatment-resistant depression (Holtzheimer et al., 2012; Schlaepfer, Bewernick, Kayser, Mädler, & Coenen, 2013). Other promising techniques such as optogenetics may provide even finer control over

the activation and deactivation of brain circuits (Dai, Brooks, & Sheinberg, 2014). Optogenetics involves injecting, into a neuron, the deoxyribonucleic acid (DNA) that encodes for activation (or deactivation) in response to light. Doing so allows controlling specific neurons with light. Although the technique is in its infancy, optogenetics holds promise for the targeted treatment of psychopathology via the control of specific neurons and brain circuits.

Effective interventions, however, need not directly target brain circuits via medication, neurofeedback, or optogenetics. Some of the most effective interventions (or preventive approaches) for externalizing problems in young children involve behavioral interventions such as parent training (Comer, Chow, Chan, Cooper-Vince, & Wilson, 2013). Effective behavioral treatments are thought to have similar effects on the brain as effective pharmacological treatments (Baxter et al., 1992; Linden, 2006; Wiswede et al., 2014), so social or behavioral interventions should not be overlooked as an important first-line treatment for behavior problems that often have fewer side effects (West, 2013) and lower relapse rates (Hollon, Stewart, & Strunk, 2006) than medication. For example, attention training (Rueda, Checa, & Cómbita, 2012; Rueda, Rothbart, McCandliss, Saccomanno, & Posner, 2005) and exercise (Drollette et al., 2014) have been shown to change the N2 and improve inhibitory control in children. Interestingly, differences in brain structure have been shown to mediate the effects of maternal depression on their children's aggression (Gilliam, Forbes, Gianaros, Erickson, Brennan, & Shaw, 2014). Thus, it is possible that parent management training reduces the child's misbehavior, in part, by helping the child build new skills that re-wire their brain. In support of this interpretation, Neville, Stevens, Pakulak, Bell, Fanning, Klein, and Isbell (2013) found that a parent training program led to improved neural processing in selective

attention and improved cognitive and behavioral functioning. Moreover, generally the earlier the intervention, the greater the likelihood of success and the lower the cost as reviewed below.

However, labeling and misidentification are potential concerns in the early identification of children at risk for developing later problems (Insel, 2014). Some children may be deemed at risk but never develop problems (false positive). Giving these children an "at risk" label could be harmful if they receive a treatment with potentially serious adverse effects (e.g., medication). As discussed above, however, some of the most effective treatments for behavior problems include behavioral interventions such as parent training and preschool that could still be beneficial for the child's development even in the case of a false positive. At the same time, sensitivity—detecting who is at risk of developing later problems—is a bigger problem today than specificity because so many people go without important and effective services until it may be too late for cost-effective interventions (Insel, 2014). The better we understand the early underlying brain mechanisms in the development of behavior problems, the greater our sensitivity and specificity to predict who will develop later problems, and the better targeting of resources to those in greatest need of services.

5.3 The Importance of Early Identification and Intervention

Early interventions such as nurse home visitation (Olds et al., 1998) and preschool (F. Campbell et al., 2014; Heckman, 2006) have been shown to be effective in reducing later criminality, and improving later income, education, and health in adulthood. Early childhood is a sensitive period for the development of self-regulation skills providing children the

capacity to respond in alternative ways to externalizing behavior. Possibly because younger children have greater neuroplasticity (Cramer et al., 2011) and a less stable self-concept (Cole, Maxwell, et al., 2001) than older children, there is greater potential for prevention and treatment of externalizing psychopathology in young children (NRC & IOM, 2009; Stormont, 2002). Although externalizing disorders are often difficult to treat, prevention efforts targeted to younger, at-risk children have been more efficacious and cost-effective in altering later trajectories (e.g., Bierman, Coie, et al., 2007), with benefit-cost ratios of early childhood interventions about 8–9 to 1 (Heckman & Masterov, 2007). For society's expected return on investment for interventions as a function of one's age, see Figure 5.1.

Despite evidence that earlier interventions would have greater cost-effectiveness than later interventions, our society's funding priorities do not reflect this knowledge. The United States spends considerably more on health care (M. M. Davis, 2013) and research (Gitterman & Hay, 2008) for adults than for children. From 1960 to 2017, the percentage of the United States domestic budget devoted to children is expected to decline from 20% to 13% (from 15% in 2006; Carasso, Steuerle, & Reynolds, 2007). From 1992 to 2009, the proportion of the budget of the National Institutes of Health devoted to research on children also declined (Gitterman & Hay, 2008).

5.4 Conclusion

In conclusion, early childhood may present an important window for understanding the early mechanisms in the development of behavior problems when the brain and behavior are

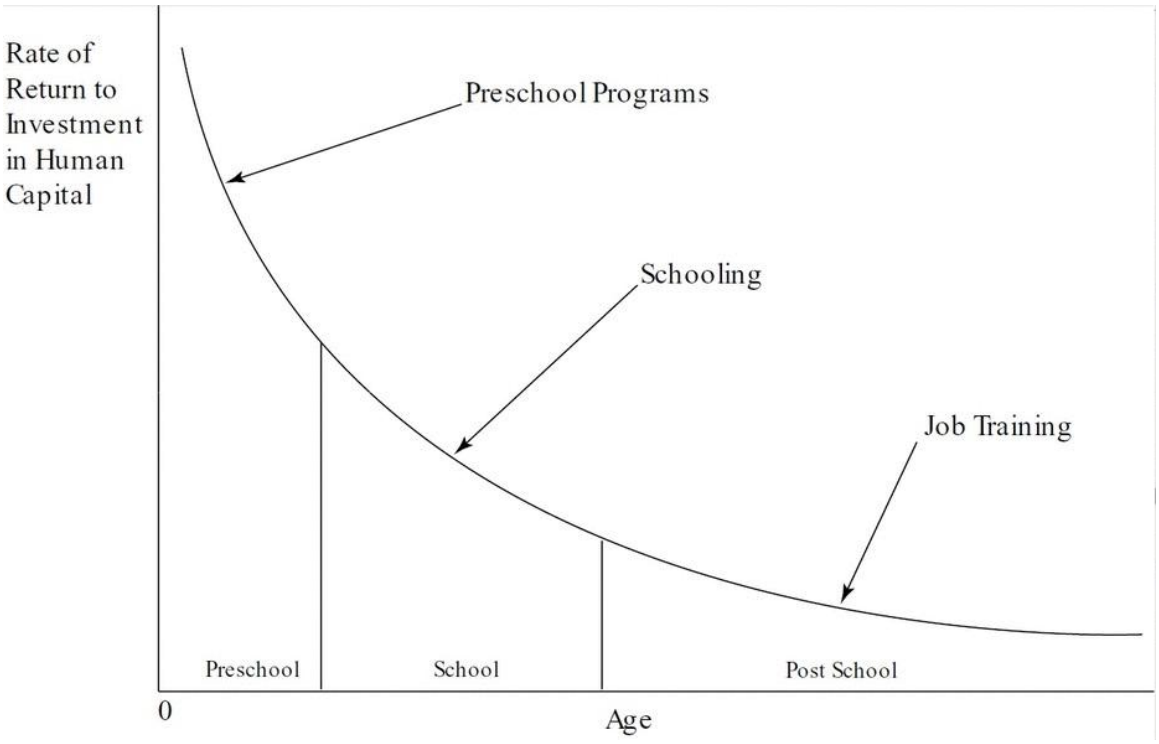

FIGURE 5.1: Estimate of the rate of return on investment in human capital as a function of one's age. Printed from Heckman and Masterov (2007) with permission from Oxford University Press.

most likely to be malleable and when interventions are most likely to be effective and cost-effective. We identified several neural markers of risk for the development of self-regulation deficits and externalizing behavior problems: smaller oddball P3b amplitudes, longer oddball P3a latencies, too small *or* too large no-go N2 amplitudes, left frontal asymmetry, and less frontal alpha power. Less frontal alpha power showed the greatest predictive value, and predicted later change in externalizing problems over time with moderate accuracy. Findings suggest that future studies should examine a new model of no-go N2 amplitudes in relation to inhibitory control whereby there may be an optimal range of inhibitory processing. Too little inhibitory processing may reflect poorer inhibitory control and too much

may reflect inefficient processing. In sum, longitudinal studies of neural functioning in relation to behavior in early childhood have the potential to greatly inform our understanding of mechanisms in the development of self-regulation and behavior problems.

It is notable, however, that we were able to examine neurodevelopmental processes on only about two-thirds of the sample because of missingness in the EEG data for various reasons. It is unclear how the one-third of children with missing EEG data may differ from the two-thirds of children we examined (apart from being younger). It is possible that we excluded a highly interesting and important subset of children with unique mechanistic processes. In order for neural measures to provide more generalizable findings and to be clinically useful, studies will have to grapple with the tradeoff between having sufficient trials or data for an adequate signal-to-noise ratio while at the same time ensuring a much lower rate of missingness than is typically observed in studies examining neurophysiological processes in childhood. Possible approaches may include requiring fewer than 10 artifact-free trials to include a participant, including incorrect trials, or using single trial analysis (Blankertz, Lemm, Treder, Haufe, & Müller, 2011). We hope that future studies examine the optimal tradeoff between these and other approaches to accommodate the population of interest that ensure the validity of neurophysiological data while maximizing generalizability and clinical utility.

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Young, S. E., Friedman, N. P., Miyake, A., Willcutt, E. G., Corley, R. P., Haberstick, B. C., & Hewitt, J. K. (2009). Behavioral disinhibition: Liability for externalizing spectrum disorders and its genetic and environmental relation to response inhibition across adolescence. *Journal of Abnormal Psychology, 118 (1)*, 117–130. DOI: 10.1037/a0014657. URL: http://psycnet.apa.org/journals/abn/118/1/117/ (cited on pp. 28, 53).

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Zelazo, P. D., Carlson, S. M., & Kesek, A. (2008). The development of executive function in childhood. *Handbook of developmental cognitive neuroscience*. Ed. by C. A. Nelson & M. Luciana. 2nd. Cambridge, MA, US: The MIT Press. Chap. 34. URL: http://psycnet.apa.org/psycinfo/2008-12890-034 (cited on p. 5).

Zhou, Q., Chen, S. H., & Main, A. (2012). Commonalities and differences in the research on children's effortful control and executive function: A call for an integrated model of self-regulation. *Child Development Perspectives, 6 (2)*, 112–121. DOI: 10.1111/j.1750-8606.2011.00176.x. URL: http://onlinelibrary.wiley.com/doi/10.1111/j.1750-8606.2011.00176.x/full (cited on p. 5).

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CONTACT INFORMATION

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LANGUAGES

English
French

RESEARCH INTERESTS

Clinical child psychology, developmental psychopathology, externalizing behavior problems, self-regulation, school readiness, developmental cognitive neuroscience

EDUCATION

Clinical Psychology Intern

Western Psychiatric Institute and Clinic, University of Pittsburgh Medical Center, Pittsburgh, PA 2015–2016
APA-Accredited Predoctoral Internship in Clinical Psychology

Ph.D.

Indiana University, Bloomington, IN 2009–2016
Ph.D., Clinical Psychology, August 2016

- Advisor: John Bates, Ph.D.
- Major: Clinical Science
- Minor: Cognitive Neuroscience
- Dissertation: Neurophysiological mechanisms in the development of externalizing behavior problems in young children

B.A.

University of Texas, Austin, TX 2005–2008
B.A., Psychology, French, December 2008

- With High Honors, Honors in Psychology, Honors in French
- Psychology Honors Thesis: *Children's Syntactic Processing of Language*
- French Honors Thesis: *Acceptability of French Borrowings in English*
- Minor: Sociology (criminology track)

AWARDS

National Institutes of Health (NIH)

- National Research Service Award (NRSA) Grant 2014–2015
- Developmental Training Grant in Integrative Developmental Process 2011–2012

National Science Foundation (NSF)

- Graduate Research Fellowship Honorable Mention 2010

Indiana Clinical and Translational Sciences Institute (CTSI)

- Predoctoral Training Grant in Translational Research 2012–2013

Society for Research in Child Development
- Student Travel Award 2011, 2015

Indiana University
- Graduate and Professional Student Organization Travel Award 2014
- College of Arts and Sciences Travel Award 2014
- Richard David Young Research Fellowship 2010, 2011
- Outstanding Instructor for Experimental Research Methods in Psychology 2011

University of Texas
- Phi Beta Kappa Society 2008–present
- Psi Chi (Psychology Honors Society) 2008–present
- Pi Delta Phi (French Honors Society) 2007–present

Boy Scouts of America
- Eagle Scout 2001

Grants

Ruth L. Kirschstein National Research Service Award (NRSA) 2014–2015
Funding Agency: NIH/NIMH
Role: Principal Investigator
Grant Title: Neurophysiological Mechanisms in the Development of Externalizing Problems (1 F31 MH100814-01A1)

Clinical and Translational Sciences Award 2012–2013
Funding Agency: NIH/NCATS
Role: Pre-doctoral trainee (PI: Anantha Shekhar, M.D., Ph.D.)
Grant Title: Indiana Clinical and Translational Sciences Institute TL1 Program (TL1 TR000162)

National Research Service Award—Institutional Training Grant 2011–2012
Funding Agency: NIH/NICHD
Role: Pre-doctoral trainee (PI: Linda Smith, Ph.D.)
Grant Title: Training Program in Integrative Developmental Process (HD-007475-17)

Publications

Peer-Reviewed Articles

[1] **Petersen, I.T.**, Hoyniak, C.P., McQuillan, M.E., & Bates, J.E. (2016). Measuring the development of inhibitory control: The challenge of heterotypic continuity. *Developmental Review, 40*, 25–71. doi: 10.1016/j.dr.2016.02.001

[2] **Petersen, I.T.**, Bates, J.E., Dodge, K.A., Lansford, J.E. & Pettit, G.S. (2016). Identifying an efficient set of items sensitive to clinical-range externalizing problems in children. *Psychological Assessment, 28(5)*, 598–612. doi: 10.1037/pas0000185

[3] Bolbecker, A.R., **Petersen, I.T.**, Kent, J.S., Howell, J.M., Westfall, D.R., O'Donnell, B.F., & Hetrick, W.P. (2016). New insights into the nature of cerebellar-dependent eyeblink conditioning deficits in schizophrenia: A hierarchical linear modeling approach. *Frontiers in Psychiatry, 7(4)*, 1–7. doi: 10.3389/fpsyt.2016.00004

[4] **Petersen, I.T.**, Bates, J.E., Dodge, K.A., Lansford, J.E. & Pettit, G.S. (2015). Describing and predicting developmental profiles of externalizing behavior problems from childhood to adulthood. *Development and Psychopathology, 27(3)*, 791–818. doi: 10.1017/S0954579414000789

[5] **Petersen, I.T.**, Bates, J.E., & Staples, A.D. (2015). The role of language ability and self-regulation in the development of inattentive–hyperactive behavior problems. *Development and Psychopathology, 27(1)*, 221–237. doi: 10.1017/S0954579414000698

- In the news:
  http://news.indiana.edu/releases/iu/2014/07/language-skills-and-behavior.shtml
  https://www.nichd.nih.gov/news/releases/Pages/081814-podcast-early-language.aspx

[6] Hoyniak, C.P., **Petersen, I.T.**, McQuillan, M.E., Staples, A.D., & Bates, J.E. (2015). Less efficient neural processing related to irregular sleep and less sustained attention in toddlers. *Developmental Neuropsychology, 40(3)*, 155–166. doi:10.1080/87565641.2015.1016162

[7] Staples, A.D., Bates, J.E., & **Petersen, I.T.** (2015). IX. Bedtime routines in toddlerhood: Prevalence, consistency, and associations with nighttime sleep. In M. El-Sheikh & A. Sadeh (Eds.), Sleep and development: Advancing theory and research. *Monographs of the Society for Research in Child Development, 80(1)*, 141–159. doi: 10.1111/mono.12149

[8] Bolbecker, A.R., Kent, J.S., **Petersen, I.T.**, Klaunig, M.J., Forsyth, J.K., Howell, J.M., Westfall, D.R., O'Donnell, B.F., & Hetrick, W.P. (2014). Impaired cerebellar-dependent eyeblink conditioning in first-degree relatives of individuals with schizophrenia. *Schizophrenia Bulletin, 40(5)*, 1001–1010. doi:10.1093/schbul/sbt112

[9] **Petersen, I.T.**, Bates, J.E., D'Onofrio, B.M., Coyne, C.A., Lansford, J.E., Dodge, K.A., Pettit, G.S., & Van Hulle, C.A. (2013). Language ability predicts the development of behavior problems in children. *Journal of Abnormal Psychology, 122(2)*, 542–557. doi: 10.1037/a0031963

[10] **Petersen, I.T.**, Bates, J.E., Goodnight, J.A., Dodge, K.A., Lansford, J.E., Pettit, G.S., Latendresse, S.J., & Dick, D.M. (2012). Interaction between serotonin transporter polymorphism (*5-HTTLPR*) and stressful life events in adolescents' trajectories of anxious/depressed symptoms. *Developmental Psychology, 48(5)*, 1463–1475. doi:10.1037/a0027471

Book Chapters

[1] Fontaine, N.M.G. & **Petersen, I.T.** (in press). Developmental trajectories of psychopathology: An overview of approaches and applications. In L. Centifanti & D. Williams (Eds.), *International Handbook of Developmental Psychopathology*. Wiley-Blackwell.

[2] Bates, J.E., Schermerhorn, A.C., & **Petersen, I.T.** (2014). Temperament concepts in developmental psychopathology. In K. Rudolph & M. Lewis (Eds.), *Handbook of Developmental Psychopathology* (3rd ed., pp. 311–329). New York: Springer.

[3] Bates, J.E., Schermerhorn, A.C., & **Petersen, I.T.** (2012). Temperament and parenting in developmental perspective. In M. Zentner & R. Shiner (Eds.), *Handbook of Temperament* (pp. 425–441). New York: The Guilford Press.

Manuscripts Under Review for Publication

Petersen, I.T., Lindhiem, O., Bates, J.E., Pettit, G.S., Lansford, J.E., & Dodge, K.A (under review). Using vertical scaling techniques to account for heterotypic continuity in psychopathology over a lengthy developmental span.

Conference Publications

Petersen, I.T., Lindhiem, O., Bates, J.E., Pettit, G.S., Lansford, J.E., & Dodge, K.A. (2016). Using vertical scaling techniques to account for heterotypic continuity in psychopathology over a lengthy developmental span. Poster presented at the University of Pittsburgh Department of Psychiatry Research Day, Pittsburgh, PA.

Petersen, I.T. (2015). Symposium Chair: Neural mechanisms of psychopathology in early childhood. Paper symposium presented at the meeting of the Society for Research in Child Development, Philadelphia, PA.

Petersen, I.T., Hoyniak, C.P., Staples, A.D., Bates, J.E., & Molfese, D.L. (2015). Neurophysiology of externalizing behavior problems in young children. In I. Petersen (Chair), *Neural mechanisms of psychopathology in early childhood*. Paper symposium presented at the meeting of the Society for Research in Child Development, Philadelphia, PA.

Petersen, I.T., Hoyniak, C.P., McQuillan, M.E., & Bates, J.E. (2015). Measuring the development of self-regulation: The challenge of heterotypic continuity. Poster presented at the biennial meeting of the Society for Research in Child Development, Philadelphia, PA.

Hoyniak, C.P., **Petersen, I.T.**, Staples, A.D., Bates, J.E., & Molfese, D.L. (2015). Development of the NoGo N2 component in relation to externalizing problems in toddlers. Poster presented at the biennial meeting of the Society for Research in Child Development, Philadelphia, PA.

Hoyniak, C.P., Bates, J.E., **Petersen, I.T.**, Yang, C-L., Darcy, I., & Fontaine, N.M.G. (2015). Atypical neural responses to vocal fear are associated with callous and unemotional behaviors in early childhood. Poster presented at the biennial meeting of the Society for Research in Child Development, Philadelphia, PA.

Petersen, I.T. (2015). Discussant: Issues in psychology and perception. Symposium presented at the Indiana University Honors Research Symposium, Bloomington, IN.

Hoyniak, C.P., **Petersen, I.T.**, Bates, J.E., Molfese, D.L., & Staples, A.D. (2014). Longer nogo N2 ERP latencies present in toddlers with sleep deficits. Symposium: The psychophysiology of self-regulation from infancy to late childhood. Symposium presented at the meeting of the Society for Psychophysiological Research, Atlanta, GA.

Hoyniak, C.P., **Petersen, I.T.**, Bates, J.E., Molfese, D.L., & Staples, A.D. (2014). ERP latencies in toddlers with sleep difficulties. Poster presented at the meeting of the Association for Psychological Science, San Francisco, CA.

Petersen, I.T., Bates, J.E., Kelsey, K.M., Hudac, C.M., Kota, S., Cortesa C., Molfese, D.L. & Staples, A.D. (2013). Less efficient neural inhibitory processing associated with externalizing behavior problems in toddlers. Poster presented at the Indiana Clinical and Translational Sciences Institute Predoctoral Programs Meeting, Indianapolis, IN.

Petersen, I.T., Bates, J.E., Kelsey, K.M., Hudac, C.M., Kota, S., Cortesa C., Molfese, D.L. & Staples, A.D. (2013). Less efficient neural inhibitory processing associated with externalizing behavior problems in toddlers. Poster presented at the National Clinical and Translational Sciences Predoctoral Programs Meeting, Mayo Clinic, Rochester, MN.

Petersen, I.T., Bates, J.E., Kelsey, K.M., Hudac, C.M., Kota, S., Cortesa C., Molfese, D.L. & Staples, A.D. (2013). N2 ERP latencies associated with self-regulation in toddlers. Poster presented at the meeting of the Association for Psychological Science, Washington, DC.

Petersen, I.T., Bates, J.E., Dodge, K.A., Lansford, J.E., & Pettit, G.S. (2013). Describing and predicting developmental profiles of externalizing problems from childhood to adulthood. Poster presented at the biennial meeting of the Society for Research in Child Development, Seattle, WA.

Petersen, I.T., Bates, J.E., Kelsey, K.M., Hudac, C.M., Kota, S., Cortesa C., Molfese, D.L. & Staples, A.D. (2013). Longer P3 latencies associated with externalizing behavior problems in young children. Poster presented at the biennial meeting of the Society for Research in Child Development, Seattle, WA.

Marks, B.T., **Petersen, I.T.**, & Bates, J.E. (2013). Prenatal testosterone exposure (2D:4D ratio) predicts aggression in young children. Poster presented at the biennial meeting of the Society for Research in Child Development, Seattle, WA.

Staples, A.D., Bates, J.E., & **Petersen, I.T.** (2013). Effects of adherence to a bedtime routine on toddlers' sleep schedule and nightly sleep. Symposium: Emerging longitudinal research linking family processes and children's sleep. Symposium presented at the biennial meeting of the Society for Research in Child Development, Seattle, WA.

Petersen, I.T., Bates, J.E., Staples, A.D., Chien, R., & Hanrahan, M. (2011). Language ability predicts development of self-regulation among toddlers. Poster presented at the biennial meeting of the Society for Research in Child Development, Montreal, Canada.

Petersen, I.T., Bates, J.E., & Staples, A.D. (2011). Interaction between parent autonomy support and child sleep in toddlers' aggression. Poster presented at the biennial meeting of the Society for Research in Child Development, Montreal, Canada.

Petersen, I.T. & Echols, C.E. (2005). Maturational constraints on second language acquisition. Poster presented at the Honors Psychology Poster Session, University of Texas, Austin, TX.

Presentations

Petersen, I.T. (2016). Treatment sequencing for childhood ADHD: A multiple randomization study of adaptive medication and behavioral interventions [Presenting findings from Pelham et al., 2016]. Project presented at Western Psychiatric Institute and Clinic (WPIC) Wide Journal Club.

Petersen, I.T. (2015). Mechanisms in the development of self-regulation. Project presented at Indiana University Dept. of Psychological and Brain Sciences Developmental Seminar.

Petersen, I.T. (2015). Mechanisms in the development of self-regulation. Project presented at Indiana University Dept. of Psychological and Brain Sciences Clinical Colloquium.

Petersen, I.T. (2015). The clinical internship process. Presented at Indiana University Dept. of Psychological and Brain Sciences Clinical Colloquium.

Vaughan, E.B. & **Petersen, I.T.** (2014). Applying for NRSA grants for predoctoral training. Presented at Indiana University Dept. of Psychological and Brain Sciences Clinical Colloquium.

Petersen, I.T., Bates, J.E., O'Donnell, B.F., & Molfese, D.L. (2013). Less efficient neural inhibitory processing associated with externalizing behavior problems in toddlers. Project presented at Mayo Clinic, National Clinical and Translational Sciences Predoctoral Programs Meeting.

Petersen, I.T., Bates, J.E., O'Donnell, B.F., & Molfese, D.L. (2013). Neurophysiological correlates of externalizing behavior problems in toddlers. Project presented at Indiana University Dept. of Psychological and Brain Sciences Cognitive Neuroscience Seminar.

Bates, J.E., & **Petersen, I.T.** (2013). Developmental models of self-regulation and adjustment. Project presented at Indiana University School of Medicine, Dept. of Otolaryngology, DeVault Otologic Research Lab.

Petersen, I.T., Bates, J.E., Kelsey, K.M., Hudac, C.M., Kota, S., Cortesa C., Molfese, D.L. & Staples, A.D. (2013). Neurophysiological mechanisms of the development of externalizing behavior problems. Project presented at Purdue University, Indiana Clinical and Translational Sciences Institute.

Petersen, I.T., Bates, J.E., Coyne, C.A., D'Onofrio, B.M., Lansford, J.E., Dodge, K.A., Pettit, G.S., & Van Hulle, C.A. (2012). The role of language ability in the development of attentional and behavioral regulation. Project presented at Indiana University Dept. of Psychological and Brain Sciences Clinical Colloquium.

Petersen, I.T., Bates, J.E., Coyne, C.A., D'Onofrio, B.M., Lansford, J.E., Dodge, K.A., Pettit, G.S., & Van Hulle, C.A. (2012). Language ability predicts the development of attention problems in children. Project presented at Indiana University Dept. of Psychological and Brain Sciences Developmental Seminar.

Petersen, I.T., Bates, J.E., Goodnight, J.A., Dodge, K.A., Lansford, J.E., Pettit, G.S., Latendresse, S.J., & Dick, D.M. (2011). Serotonin transporter gene polymorphism moderates the effect of stressful life events on trajectories of anxious/depressed symptoms. Project presented at Indiana University Dept. of Psychological and Brain Sciences Developmental Seminar.

Petersen, I.T., Bates, J.E., Staples, A.D., Chien, R., & Hanrahan, M. (2010). Language ability predicts inhibitory and effortful control among toddlers. Project presented at Indiana University Dept. of Psychological and Brain Sciences Developmental Seminar.

Clinical Experience

Supervisory Role

Parent Behavior Training (Fall 2012–Spring 2015)

Duties: Supervised junior-level graduate students individually and in group settings following an empirically-supported parent behavior training protocol to treat children's disruptive behavior disorders.

Site: Indiana University—Dept. of Psychological and Brain Sciences—Bloomington, IN

Supervisor: John Bates, Ph.D.

Evidence-Based Clinical Supervision Course (Spring 2014)
Duties: Read and discussed evidence-based information on the research, theory, and practice of supervision. Course content included practical skills for the implementation of evidence-based supervision as well as diversity and ethical issues relevant to supervision.
Site: Indiana University—Dept. of Psychological and Brain Sciences—Bloomington, IN
Supervisor: Cara Lewis, Ph.D.

Alternative Alcohol Intervention Program (Fall 2012–Summer 2013)
Duties: Trained and supervised junior-level students in their delivery of brief interventions and psychoeducation in an alcohol diversion program. Provided recommendations when students were competent to advance to individual therapists.
Site: Indiana University—Office of Student Affairs—Bloomington, IN
Supervisor: Walter Keller, Ph.D.

Evidence-Based Psychosocial Intervention Consultation (Fall 2011–Spring 2013)
Duties: Consultation with other doctoral students regarding evidence-based approaches to assessment and treatment. Met biweekly in group setting.
Site: Indiana University—Dept. of Psychological and Brain Sciences—Bloomington, IN
Supervisor: Cara Lewis, Ph.D.

Clinician Role

Dual Diagnosis Inpatient Services (Summer 2016)
Duties: Conducted psychological evaluations and completed intensive therapy using a Motivational Interviewing (MI) approach for individuals with substance abuse problems and other serious mental health difficulties.
Site: University of Pittsburgh School of Medicine—Western Psychiatric Institute and Clinic—Pittsburgh, PA
Supervisor: Antoine Douaihy, M.D.

Psychotherapy Training Clinic (Fall 2015–Summer 2016)
Duties: Conducted longitudinal, one-on-one therapy using various treatment modalities, including Cognitive Behavioral Therapy (CBT), Exposure Therapy, and Interpersonal Psychotherapy (IPT).
Site: University of Pittsburgh School of Medicine—Western Psychiatric Institute and Clinic—Pittsburgh, PA
Supervisor: Jay Fournier, Ph.D.

Diagnostic Evaluation Center (Spring 2016)
Duties: Evaluated patients presenting to the WPIC psychiatric emergency room, determined preliminary diagnoses and disposition with attending psychiatrists, and arranged for necessary treatment arrangements.
Site: University of Pittsburgh School of Medicine—Western Psychiatric Institute and Clinic—Pittsburgh, PA
Supervisor: Christopher Parada, M.A.

Science and Practice for Effective Children's Services (Winter 2016–Spring 2016)

Duties: Provided an evidence-based treatment for caregivers and their children dealing with child physical abuse (Alternatives for Families: A Cognitive Behavioral Therapy; AF-CBT). Provided behavioral health consultations and assisted in implementing a behavioral and mental service in a local, primary care practice in McKeesport, PA.
Site: University of Pittsburgh School of Medicine—Western Psychiatric Institute and Clinic—Pittsburgh, PA
Supervisor: David Kolko, Ph.D.

Obsessive-Compulsive Disorder Intensive Outpatient Program for Children and Adolescents (Spring 2016)

Duties: Co-facilitated an Exposure and Response Prevention (ERP) group for children with OCD.
Site: University of Pittsburgh School of Medicine—Western Psychiatric Institute and Clinic—Pittsburgh, PA
Supervisors: Shoshanna Shear, M.D., Amy Kelly, M.D.

Matilda H. Theiss Child Development Center (Fall 2015–Winter 2016)

Duties: Helped teachers in a therapeutic preschool manage classroom misbehavior, conducted functional behavioral assessments (FBAs) to make recommendations to teachers, and worked with families in Parent Management Training (PMT).
Site: University of Pittsburgh School of Medicine—Western Psychiatric Institute and Clinic—Pittsburgh, PA
Supervisor: Kimberly Blair, Ph.D.

Family Therapy Training Center (Fall 2015–Winter 2016)

Duties: Provided structural family therapy treatment to families experiencing a wide range of psychiatric disorders, phase-of-life problems, and problems in coping with acute or chronic stressors.
Site: University of Pittsburgh School of Medicine—Western Psychiatric Institute and Clinic—Pittsburgh, PA
Supervisor: Leonard Woods, LCSW

Behavioral Sleep Medicine (Winter 2016)

Duties: Conducted assessments of patients with insomnia and other circadian rhythm disorders, and implemented Brief Behavioral Treatment for Insomnia (BBTI), a modified version of CBT-I.
Site: University of Pittsburgh School of Medicine—Western Psychiatric Institute and Clinic—Pittsburgh, PA
Supervisors: Brant Hasler, Ph.D., Daniel Buysse, M.D.

ADHD Across the Lifespan Clinic (Fall 2015)

Duties: Worked with children with ADHD and their families in intervention, and co-led skills groups for adults and college students with ADHD.
Site: University of Pittsburgh School of Medicine—Western Psychiatric Institute and Clinic—Pittsburgh, PA
Supervisor: Aaron Jennings, LCSW

Parent Behavior Training (Fall 2010–Spring 2015)

Duties: Trained parents to manage their children's oppositional and defiant behavior with principles from social learning perspective using Fleischman's family-based parent behavior training protocol. Conducted school and home observations as needed. Maintained a weekly caseload of 1–2 families. Videotaped each session for weekly individual, group, and online supervision. Collaborated with community mental health service providers, physicians, and school teachers.
Site: Indiana University—Dept. of Psychological and Brain Sciences—Bloomington, IN
Supervisor: John Bates, Ph.D.

Child Mood Disorders Clinic (Spring 2013–Fall 2013)
Duties: Provided cognitive behavioral therapy treatment to children with depression, anxiety, and other mood disorders, in addition to co-morbid externalizing problems. Maintained a weekly caseload of 3–5 patients. Received weekly supervision.
Site: Riley Hospital for Children (Dept. of Child and Adolescent Psychiatry)—Indianapolis, IN
Supervisor: Ann Lagges, Ph.D.

Child Neuropsychology Assessment (Summer 2012–Fall 2012)
Duties: Conducted child and adolescent neuropsychology assessments for ADHD and learning disorders to help make treatment recommendations. Trained in testing, administration/scoring, behavioral observations, and preliminary case formulations. Received weekly supervision.
Site: Riley Hospital for Children (Dept. of Child and Adolescent Psychiatry)—Indianapolis, IN
Qualified Psychometric Technician: WISC-IV, K-BIT-2, WJ-III-Ach, SCWT, CIT, CCPT, CVLT-C, WRAML-2, VMI, TAT, SC, WASI-II (abbreviations defined in "Assessments" section)
Supervisor: William Kronenberger, Ph.D.

Alternative Alcohol Intervention Program (Fall 2011–Summer 2013)
Duties: Conducted brief motivational interviewing and behavioral intervention strategies for students struggling with alcohol and drug misuse and associated negative academic and legal ramifications. Maintained a weekly caseload of 7–10 individual clients. Received weekly supervision.
Site: Indiana University—Office of Student Affairs—Bloomington, IN
Supervisor: Walter Keller, Ph.D.

Child-Informed Divorce Mediation (Fall 2009–Spring 2010)
Duties: Served as a child consultant to bring the children's perspective into divorce mediation and help parents consider their children's needs. Conducted child and family assessments, and delivered feedback to parents in divorce mediation. Received weekly supervision.
Site: Indiana University—Dept. of Psychological and Brain Sciences—Bloomington, IN
Supervisor: Amy Holtzworth-Munroe, Ph.D. and Brian D'Onofrio, Ph.D.

Research Experience

Doctoral Student, Social Development Laboratory (Fall 2009–present)
Indiana University—Dept. of Psychological and Brain Sciences
Advisor: John Bates, Ph.D.
Dissertation: "Neurophysiological mechanisms in the development of externalizing behavior problems in young children"

Experience: Used longitudinal designs to examine the mechanisms involved in the development of self-regulation, conduct problems, and maladjustment. Developed system for sharing and merging data across sites as part of multi-site NIH grant. Conducted EEG assessments with 2–3-year-old children for dissertation.

Psychology Honors Project and Thesis (Spring 2008–Fall 2008)
University of Texas—Dept. of Psychology
Advisor: Charles Holahan, Ph.D. and Catharine Echols, Ph.D.
Thesis: "The role of maturational constraints in the syntactic processing of language by children and adults: a study of the less-is-more hypothesis with American Sign Language"
Experience: Designed and conducted honors project examining how children acquire second languages better than adults.

French Honors Project and Thesis (Spring 2008–Fall 2008)
Indiana University—Dept. of French & Italian
Advisor: Carl Blyth, Ph.D.
Thesis: "Des facteurs linguistiques et sociolinguistiques de l'acceptabilité des emprunts français en anglais" (Linguistic and sociolinguistic factors of acceptability of French borrowings in English)
Experience: Designed and conducted honors project examining the characteristics of French words that lead to their usage in English.

Research Assistant, Children's Research Laboratory (Spring 2006–Spring 2007)
University of Texas—Dept. of Psychology
Advisor: Catharine Echols, Ph.D.
Experience: Conducted longitudinal, cross-cultural study on language acquisition in infants (9, 13, 18 mos.). Studied the effects of priming on elicited sentence structure in 4 year olds.

Teaching Experience

Course Instructor
- Methods of Experimental Psychology (Fall 2011)

Guest Lecturer
- "Psychological Disorders of Childhood", *Abnormal Psychology* (Fall 2009)
- "ADHD", *Clinical Neuroscience* (Fall 2011, Spring 2011, Fall 2014)
- "Genes and Behavior", *Clinical Neuroscience* (Spring 2011)

Assistant Instructor
- Behavioral Disorders of Childhood and Adolescence (Fall 2009)
- Abnormal Psychology (Fall 2009, Spring 2010, Fall 2010)
- Clinical Neuroscience (Spring 2010)

Other
- Preschool Teacher (Spring 2009–Summer 2009)
- English Teacher at Professional French Business School (Fall 2007)
- After-School Elementary Teacher (Fall 2006–Spring 2007)

Editorial Experience

Ad hoc reviewer:
Development and Psychopathology
Developmental Neuropsychology

Developmental Psychology
Early Education and Development
Emotion
Infant and Child Development
Journal of Adolescent Health
Journal of Child Psychology and Psychiatry
Research in Developmental Disabilities
Translational Issues in Psychological Science

Workshops

Statistics Training

- Structural Equation Modeling: Advanced Longitudinal Modeling (2010, University of Kansas), Todd Little, Ph.D.
- Factorial Invariance in Multiple Group and Longitudinal Models (2011, University of Michigan), Todd Little, Ph.D.
- The Bayesian Perspective in the Context of Large Scale Assessments (2011, Indiana University), David Kaplan, Ph.D.
- Interpreting Interaction Effects and New Perspectives on Interaction Analysis (2011, Indiana University), James Jaccard, Ph.D.

EEG/ERP Training

- ERP Data Collection, Processing, and Analysis with Children (2011, University of Nebraska), Dennis Molfese, Ph.D.
- EEG Workshop on Artifact Detection, Rejection, and Removal (2012, Indiana University), William Hetrick, Ph.D. and Paul Kieffaber, Ph.D.

Clinical Training

- Fostering a Safe Base for Trainees to Learn, Develop and Acquire Competence (2016, Western Psychiatric Institute and Clinic), Paula Ravitz, M.D.
- Unified Protocol for Transdiagnostic Treatment of Emotional Disorders (2016, Western Psychiatric Institute and Clinic), Shannon Sauer-Savala, Ph.D.
- Structured Clinical Interview for DSM-IV-TR Axis I Disorders (2012, Indiana University), Cara Lewis, Ph.D.
- Child Inclusive Dispute Resolution (2009, Indiana University), Jennifer McIntosh, Ph.D.

Formal Ethics Training

- Responsible Conduct of Research Series: Collaborative Science (2013), Office of Research Ethics, Education & Policy, Indiana University
- Responsible Conduct of Research Series: Mentorship (2013), Office of Research Ethics, Education & Policy, Indiana University
- Responsible Conduct of Research Series: Authorship and Publications (2013), Office of Research Ethics, Education & Policy, Indiana University

Professional Experience

Village Church Child and Family Development Center, Prairie Village, KS

Preschool Teacher 2009

- Taught 4-5 year olds at a preschool, prepared lessons

IMS Business School, Nantes, France

English Teacher 2007

- Prepared lessons, taught English classes to native French students in a professional business school

Community New Start, Austin, TX

After-School Teacher 2006–2007

- Provided under-privileged elementary school children with an after-school program for tutoring, mentoring, recreational activities, and spiritual awareness

Professional Activities	Clinical Science Student Representative, Indiana University	2014–2015
	Advisory Board, Workshop in Methods, Indiana University	2012–2015
Volunteering	Basketball Coach for 3rd Graders	2014
Affiliations	Society for a Science of Clinical Psychology	2015
	Society for Research in Child Development	2010
	American Psychological Association	2010
	Association for Psychological Science	2009
	American Association for the Advancement of Science	2009
	Phi Beta Kappa Society	2009
	Psi Chi (Psychology Honors Society)	2008
	Pi Delta Phi (French Honors Society)	2007

Assessments

Beery-Buktenica Developmental Test of Visual-Motor Integration (VMI)
California Verbal Learning Test–Children's Version (CVLT-C)
Conners' Continuous Performance Test II (CCPT)
Counting Stroop Interference Test (CIT)
Differential Ability Scales-II (DAS-II)
Kaufman Brief Intelligence Test-2 (K-BIT-2)
Peabody Picture Vocabulary Test-4 (PPVT-4)
Stroop Color and Word Test (SCWT)
Tendler Sentence Completion Test (SC)
Thematic Apperception Test (TAT)
Wechsler Abbreviated Scale of Intelligence-II (WASI-II)
Wechsler Intelligence Scale for Children-IV (WISC-IV)
Wide Range Assessment of Memory and Learning-2 (WRAML-2)
Woodcock-Johnson III Tests of Achievement (WJ-III-Ach)
Woodcock-Johnson III Tests of Cognitive Abilities (WJ-III-Cog)

Technical Skills

Neurophysiological Techniques: Electroencephalography (EEG), Event-related potentials (ERPs)

Statistical Analysis: Multilevel Modeling/Hierarchical Linear Modeling (MLM/HLM), Structural Equation Modeling (SEM), Moderated Multiple Regression, Principal Components Analysis (PCA), Latent Growth Curve Modeling (LGCM), Longitudinal Mediation Analysis, Cross-Lagged Autoregressive Panel Modeling, Latent Change/Difference Score Modeling (LCS/LDS), Autoregressive Latent Trajectory Modeling (ALT), Multiple Imputation, Item Response Theory (IRT)

Statistical Software: R, Mplus, SPSS, SAS, OpenMx, AMOS, MATLAB, ERP PCA Toolkit, Amelia

Computer Applications: Net Station, TEX (LATEX, BIBTEX, Beamer), GitHub, most common productivity packages for Windows

Operating Systems: Microsoft Windows, Apple OS, Linux

Research Supervision

People

- Undergraduate research assistants: 25
- Graduate assistants: 3
- Graduate students: 2

Theses, Grants, and Publications

- McNair Project and Thesis: 2
- Honors Project and Thesis: 3
- Pre-doctoral NSF grant: 1
- Article submission to peer-reviewed journal: 2
- Article published in peer-reviewed journal: 1

Activities

- Trained others in behavioral coding of parent–child interaction tasks
- Trained others in data analysis, including Pearson correlation, multiple regression, moderated multiple regression, principal components analysis, multilevel modeling, structural equation modeling, and longitudinal data analysis
- Trained others in R software and coding for data management, merging, cleaning, calculating composites, and statistical analysis
- Developed and trained others in data management system for multi-site NIH study with double data entry
- Trained others in open and collaborative science with version control using GitHub
- Trained others in EEG, including use of technical equipment, net placement on young children, data processing, interpretation, and data analysis
- Worked closely with junior graduate student on pre-doctoral NSF grant submission that built on my dissertation study; grant was awarded
- Published in a peer-reviewed journal with junior graduate student using EEG data from my dissertation study
- Submitted a manuscript to a peer-reviewed journal with a former undergraduate research assistant whose Honors project I supervised